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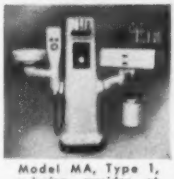

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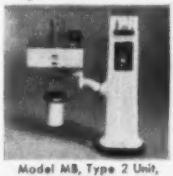

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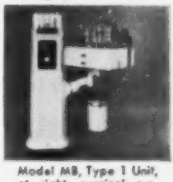

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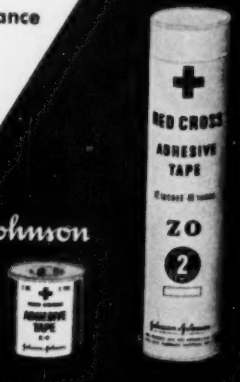

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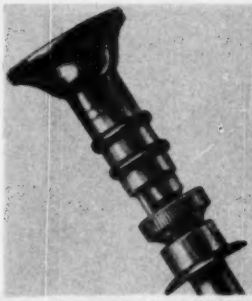

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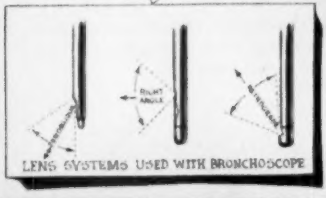

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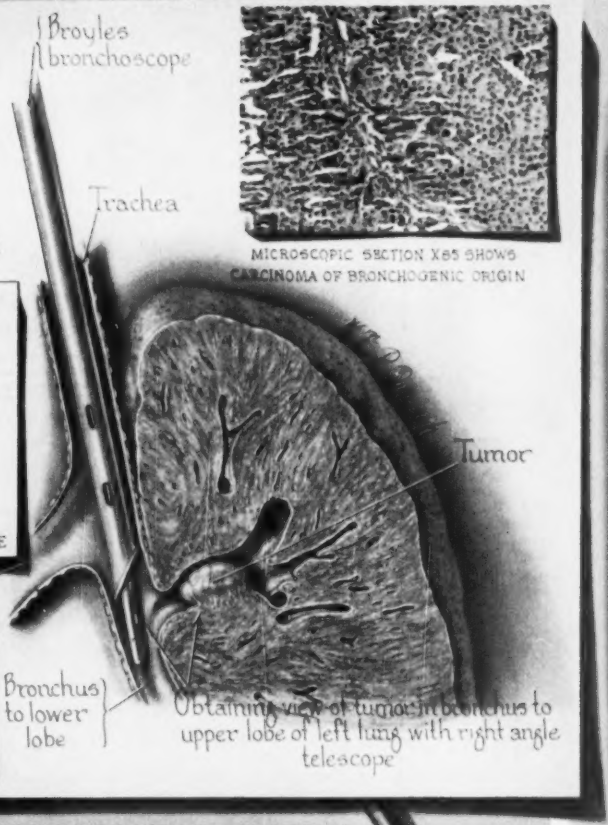

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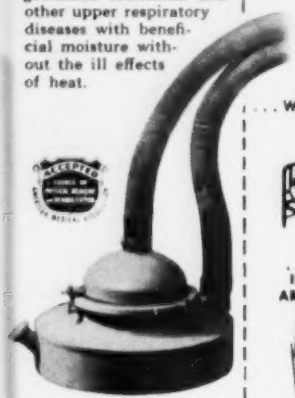

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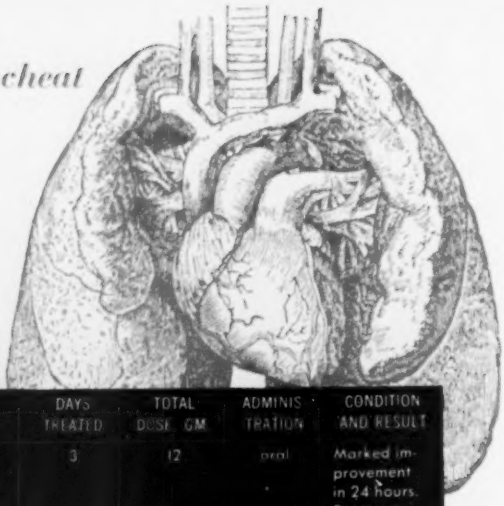

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1. Welch, H.; Hendricks, E. D.; Price, C. W., and Randall, W. A.: J. A. Ph. A. (Sc. Ed.) 39:185 (Apr.) 1950.
2. Knight, V.: New York State J. Med. 50:2173 (Sept. 15) 1950.

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CONTENTS

PAGE

Symposium on the Histology and Pathogenesis of Otosclerosis:

Fissular Region of the Otic Capsule in Relation to Otosclerosis. BARRY J. ANSON, PH.D. (MED. SC.), CHICAGO 843

Incidence, Location and Extent of Otosclerotic Lesions. STACY R. GUILD, PH.D., BALTIMORE 848

Otosclerosis: Hypothesis of Its Origin and Progress. DOROTHY WOLFF, PH.D., NEW YORK 853

Influence of Systemic and Local Factors on the Development of Otosclerosis. J. R. LINDSAY, M.D., CHICAGO 868

Postnatal Rebuilding and Otosclerotic Bone Formation in the Region of the Otic Capsule. T. H. BAST, PH.D., MADISON, WIS. ... 882

Critical Evaluation of Antihistaminic Drugs in the Common Cold. NOAH D. FABRICANT, M.D., CHICAGO 888

Classification of Chronic Diseases of Nose and Accessory Sinuses. NOAH FOX, M.D., AND LEONARD M. NILES, M.D., CHICAGO 900

Streptomycin Therapy in Oral Tuberculosis. H. OPPENHEIM, M.D.; C. S. LIVINGSTON, M.D.; J. W. NIXON, D.D.S., AND C. D. MILLER, D.M.D., OUTWOOD, KY. 910

Tympanomeatal Membrane in the Fenestration Operation: Its Relation to Bony Closure of the Fenestra. SAMUEL ROSEN, M.D., NEW YORK 930

Use of Vinegar as an Antibiotic in the Treatment of Chronic Middle Ear Disease. IRVING L. OCHS, M.D., ANNAPOLIS, MD. 935

CASE REPORTS:

Rhabdomyosarcoma of the Maxillary Antrum: Seven Year Survival Following Surgical Excision and Radiation Therapy. PETER N. PASTORE, M.D.; PHILIP F. SAHYOUN, M.D., AND FREDERICK B. MANDEVILLE, M.D., RICHMOND, VA. 942

CLINICAL NOTES:

Histamine Treatment of Bell's Palsy: Report of Five Cases. G. L. LOOMIS, M.D., WINONA, MINN. 948

An Operation for Relief of Stenosis of the Stoma Following Total Laryngectomy. JOHN MARQUIS CONVERSE, M.D., NEW YORK .. 950

A Skin Resistance Audiometer. JOHN A. KIRCHNER, M.D., NEW HAVEN, CONN., AND HARRY H. HAGGART, M.D., CINCINNATI 952

Infraction of the Inferior Turbinates. OTTO L. E. DE RAADT, M.D. (LEIDEN), TJIMAHI, JAVA 956

Tooth Guard for Use with Davis Mouth Gag. RALPH S. LLOYD, D.D.S., AND RHEIM M. JONES, M.D., BALTIMORE 960

A New Nasal Splint. IRVING B. GOLDMAN, M.D., NEW YORK 962

PROGRESS IN OTOLARYNGOLOGY. Summaries of the Bibliographic Material Available in the Field of Otolaryngology for 1947 and 1948:

Plastic Surgery. LYNDON A. PEER, M.D.; JOHN C. WALKER, M.D., AND JOHN VAN DUYN, M.D., NEWARK, N. J. 964

SPECIAL REPORTS:

Hearing Aids Accepted by the Council on Physical Medicine and Rehabilitation, the American Medical Association, as of November 1, 1950 993

ABSTRACTS FROM CURRENT LITERATURE 998

BOOK REVIEWS 1011

SPECIAL NOTICE 1011

DIRECTORY OF OTOLARYNGOLOGICAL SOCIETIES 1012

CONTENTS OF VOLUME 52 i

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ARCHIVES OF OTOLARYNGOLOGY
VOLUME 52 DECEMBER 1950 NUMBER 6

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SYMPOSIUM ON THE HISTOLOGY AND PATHOGENESIS OF OTOSCLEROSIS

FISSULAR REGION OF THE OTIC CAPSULE IN RELATION TO OTOSCLEROSIS

BARRY J. ANSON, Ph.D.(Med.Sc.)
CHICAGO

IN AN investigation based on an examination of more than 300 series of sections, covering specimens from the 6 mm. embryo to the 70 year old adult, the development of the temporal bone and its adult histological structure have been studied, with particular reference to pathological changes in the fissular region of the otic capsule.

In this report, which must be brief, the development of the fissular region and the neighboring portions of the otic capsule, the normal adult structure of the same regions and these pathological alterations which occur in and around the fissula which result in one form of deafness will be discussed.

The adult temporal bone is "petrous" only where it serves as a bed for the membranous labyrinth. The petrous part is only that portion which houses the otic labyrinth. Marrow and air spaces occupy much of the remaining portion. They are late fetal additions to the primordial otic capsule.

Despite its relative density, the capsule is crossed by four fissures of perilymphatic nature, namely, the vestibular and cochlear aqueducts, the fossula post fenestram and the fissula ante fenestram. From the present standpoint, the most important of these is the fissula, named for its position anterior to the fenestra vestibuli (oval window). The fissula

Contribution no. 531 from the Department of Anatomy, Northwestern University Medical School.

Detailed illustrated accounts of the observations reported herein have already been published (Ann. Otol., Rhin. & Laryng. **56**:957-985 [Dec.] 1947; **57**:103-128 [March] 1948; **57**:603-632 [Sept.] 1948).

This paper is the first in a "Symposium on the Histology and Pathogenesis of Otosclerosis," held at the meeting of the Otosclerosis Study Group, Division of the Committee on Conservation of Hearing, in connection with the Fifty-Fourth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Chicago, Oct. 8, 1949.

is consequential because it lies at the core of the so-called site of predilection of otosclerosis.

The fissula ante fenestram begins externally on the medial wall of the tympanic cavity. It ends internally at the junction of the vestibule and the scala vestibuli. The fissula is a constant anatomic feature. Although typically it is filled with a moderately vascular fibrous tissue, it is sometimes occupied by a large mass of cartilage, or by bone of sclerotic type. For this reason the morphogenesis of the fissular region is worthy of detailed study.

DEVELOPMENT AND NORMAL STRUCTURE OF THE FISSULA ANTE FENESTRAM

The fissula makes its appearance in the fetus of 9 weeks (34 mm.) as an anteriorly directed outpushing of the periotic connective tissue of the vestibule. In the fetus of 10 weeks (50 mm.) there occurs a dissolution of that portion of the precartilage of the primitive capsule which immediately surrounds the periotic projection—in just the way that the same tissue is removed to permit enlargement of any part of the otic labyrinth. Cartilage is formed, then locally destroyed. The fissula gradually widens and lengthens, reaching the opposite, or tympanic, surface of the capsule in the fetus of 13 weeks (75 mm.). The process of burrowing requires five weeks, and the fissular tract is distinct in the 14 week (100 mm.) fetus. In this way a perilymphatic channel is produced, complete from the inner to the middle ear.

By the fifth month of prenatal life (in the fetus of 21 weeks, or 183 mm.) the fissula has attained maximum normal size. Concurrently, the cochlea, the vestibule, the semicircular ducts, the otic capsule and the ossicles also reach full adult dimensions. The remaining prenatal months and all the years of infancy, childhood and manhood are available, so to speak, for histological maturation or for genesis of pathological tissue.

At this stage (midterm fetus) the cartilage of the primordial capsule remains as a tube within which is lodged the fibrous tissue derived from the original periotic extension. This cartilage is continuous with a chondral rim bordering the vestibular window. This continuity is maintained even in advanced age. At the tympanic extremity of the fissula there is similar continuity—later to be interrupted. Perichondrial bone covers the capsule. Between this platelike bone on the periphery and the cartilage enclosing the fissula, at the core of the capsule, osseous tissue of an unusual type is being formed through rapid calcification and ossification. This converted cartilage is intrachondral bone, composed of so-called cartilage islands.

The cartilage has been almost completely removed by the 23 week (210 mm.) stage. It is represented only by a thin chondral or perichondrial layer. This pellicle lines the newly formed bone which has spread

to form an uninterrupted wall for the fissula. The fibrous content remains unchanged.

In mature form the fissula passes inward from a lateral orifice, which opens on the submucosal tissue of the tympanic cavity. Turning at a right angle, toward the vestibular window and away from the cochlea, the fissular tract descends as it passes to an opposite, medial, orifice. This medial, inner, orifice opens on the periotic tissue of the vestibule. The cartilage which lines the wall of the vestibule remains continuous with that of the fenestra; usually in the adult it does not reach the tympanic orifice. Usually the fissula approaches, but does not terminate in, the vestibular window.

In some specimens an auxiliary, or fenestral, opening occurs (as may be found in specimens from all age groups). In such instances the connective tissue of the fissula is directly continuous with the annular ligament of the stapes—or, in terms of space rather than content, the fissula opens into the vestibular window. When present, the fenestral opening is established in the fetus; it appears as a deep oblique cleft in the capsular wall and extends without interruption from the wall of the vestibule upward and outward, across the rim of the vestibular window to the tympanic cavity. Now, were the cartilage lining of the fissula to undergo new growth, its spreading tissue could invade the vestibular window by way of a channel ready made. Therefore, to cause a deforming pathological process, there is merely required chondromatous growth or replacement of the latter by sclerotic bone.

PATHOLOGICAL CONTENTS OF THE FISSULA

The "space" of the fissula, that is, the capsular fault occupied normally by connective tissue, possesses typically the form of the letter C. Usually it is compressed, to appear bladelike; in some instances the fissula extends upward (that is, cranialward) beyond the level of the tympanic orifice; not infrequently the fissula is capacious throughout its entire length.

A large fissular tract seemingly invites histological activity. Cartilage, produced by the normally quiescent perichondrium of the fissular rim, may come to occupy part, or it may obliterate all, of the enlarged fissula ante fenestram, as if the latter were a structural defect.

In anticipation of conclusions based on observations next to be presented, it may be said that, although otosclerotic bone has been found in various portions of the otic capsule, the commonest site of occurrence is the region of the fissula ante fenestram. This is the area, too, in which masses of abnormal cartilage, or chondromas, are most frequently seen. Of course, simple masses of residual cartilage occur elsewhere in the otic capsule; they, however, disappear at the time of birth or during

early postnatal years. These transitory nodules or seams are found at the base of the styloid process, at the petrosquamous suture, at the subarcuate fossa, around the inferior margin of the cochlear (round) window and near the internal acoustic meatus.

It is only in the fissular region that cartilage is formed anew, postnatally, to encroach on a space which in most instances (that is, normally) remains filled with connective tissue. In the fissula the abnormal mass develops from the chondral rim, already described and pictured, which intervenes between the fissular connective tissue and the surrounding shell of bone. The new cartilage is histologically different from that of the parent tissue; depending on the stage encountered, it is either actively spreading or is preparing to undergo initial ossification. In almost every instance the cartilage occurs in a fissula which is unusually large, as if it were reducing the size of the fissular cleft in the bone. Such a chondroma may occupy either end of the fissula, the fenestral orifice which opens into the stapedial joint, the cupola-like space which sometimes occurs cranial to the level of the tympanic opening, or even the entire space of the fissula.

Regularly, the margin of such a chondral mass slowly undergoes ossification, bone replacing cartilage by a familiar series of steps, to form a gnarly lump. Sometimes the process does not stop when an osseous nodule has been produced; rebuilding continues, further excavation being accompanied with new deposition, with resultant obliteration not only of the fissular connective tissue but also of the quiescent cartilage of the original fissular rim and the surrounding endochondral and intrachondral bone of the otic capsule. In advanced stages the otosclerotic bone reaches the vestibular window and takes the place of the normal tissue. This is the terminal stage usually described in the literature.

When these circumstances are put together into a series of developmental steps, it seems that the natural sequence is the following: (1) closure, partial or entire, of a large (atypical) fissula by fetal or infantile growth of cartilage—the latter tissue derived from the perichondrial layer which always borders the chondral rim of the fissular tract; (2) gradual osseous replacement of the chondroma, a process which in some cases continues to a pathological degree—replacing the normal tissues of the entire fissular region with sclerotic bone.

SUMMARY AND CONCLUSIONS

Factual.—In the antefenestral region of the otic capsule, that is, in the area between the vestibular window and the cochlea, the structure is exceptional in being crossed by a channel (termed the fissula ante fenestram).

The fissula is formed through invasion of the primordial cartilaginous capsule by a tongue of periotic tissue; maturing, the latter becomes the

connective tissue which regularly occupies the fissular tract of the adult ear.

During the process of ossification of the capsule cartilage persists as the lining of the fissula, external to which, as early as the midterm stage, bone forms a completely investing osseous shell.

Typically, the fissular channel is C-shaped in outline and narrow or cleftlike and is occupied by connective tissue.

Frequently in the advanced fetus, in the infant and in the child the fissula is capacious, the space being cylindrical rather than bladelike. In such instances a cartilage mass, or chondroma, almost invariably occupies part or all of the fissula; in some instances it remains confined to the enlarged cranial part; in others it occupies the entire fissular tract. The chondroma may reach the stapes directly when an auxiliary (fenestral) orifice occurs.

This neoplastic cartilage is derived from the persistent chondral lining of the fissula.

Usually the chondromatous mass is gradually reduced by being converted, from the periphery, into intrachondral bone. In relatively rare instances, however, the replacement is rapid and is carried out through the production of a vascular sclerotic type of osseous tissue. Being invasive, this new vascular bone displaces normal surrounding tissue, to form part of the rim of the vestibular window and, as a consequence, to impinge on the base of the stapes.

In no other portion of the otic capsule is there encountered like evidence of histological instability—a circumstance which renders the fissular region subject to pathological change, the vestibular window vulnerable to invasion and the stapes subject to fixation within its fenestra.

Hypothetical.—It would appear that the activity by which cartilage is produced within the fissula represents an attempt to close an abnormally large cleft in the antefenestral area of the otic capsule.

It would seem, further, that the ultimate formation of sclerotic bone is a natural second step in the same process. Having been formed, the vascular bone spreads beyond the area of the original capsular defect.

INCIDENCE, LOCATION AND EXTENT OF OTOSCLEROTIC LESIONS

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OTOSCLEROSIS is not a rare disease in Caucasians. Nothing is known about its incidence in the yellow races of mankind or in any of the unmixed black races. In the American Negro, otosclerosis occurs much less frequently than it does in the white population of this country. The disease, so far as is known, does not occur in any species except Homo sapiens.

Otosclerosis is primarily a disease of bone; the effect of the disease on hearing is secondary to the location and the extent of the bony lesion.[1] If the figures that I reported to this Study Group at its 1943 meeting [1a] hold essentially true as larger collections of material are prepared and studied, approximately one eighth of all white females, including girls 5 years of age and older, and about one fifteenth of all white males of the corresponding age group have otosclerotic lesions.

The incidence of clinical otosclerosis is far below that of the histological, because in about seven eighths of the ears with otosclerotic lesions

From The Johns Hopkins University School of Medicine.

This paper is the second in a "Symposium on Histology and Pathogenesis of Otosclerosis," held at the meeting of the Otosclerosis Study Group, Division of the Committee on Conservation of Hearing, in connection with the Fifty-Fourth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Chicago, Oct. 8, 1949.

In the presentation of this paper, 11 lantern slides were used to illustrate the points discussed. Each was a photomicrograph of a histological section of a human ear with otosclerosis. All the photomicrographs have been previously published in my paper "Histologic Otosclerosis" (Ann. Otol., Rhin. & Laryng. **53**:246-266 [June] 1944) or in my paper "Early Stages of Otosclerosis" (Arch. Otolaryng., **12**:457-483 [Oct.] 1930). Because these sources are readily available to most readers, republication of the illustrations does not seem justified and has not been requested.

1. (*a*) Guild, S. R.: Histologic Otosclerosis, Ann. Otol., Rhin. & Laryng. **53**:246-266 (June) 1944. (*b*) Engström, H.: Über das Vorkommen der Otosklerose, nebst experimentellen Studien über chirurgische Behandlung der Krankheit, Acta oto-laryng., 1940, supp. 43, pp. 1-153. (*c*) Nylén, B.: Histopathological Investigations on the Localization, Activity and Extent of Otosclerotic Foci, Upsala läkaref. förh. **54**:1-52, 1949.

the condition gives rise to no symptoms, and the presence of the disease is never suspected unless serial histological sections of the temporal bones are made and studied with a microscope. Only in about one eighth of the ears with histologically demonstrable otosclerosis are the location and the extent of the osseous lesion such that impaired hearing results. There may be occasional exceptions, but in general otosclerosis does not affect hearing acuity until, or unless, ankylosis of the stapedio-vestibular articulation occurs.

In other words, to cause impaired hearing an otosclerotic lesion must invade, and replace with its peculiar kind of new bone, some portion of the articular cartilage and the adjacent part of the annular ligament. The mere presence of an otosclerotic lesion near or in contact with the annular ligament does not interfere with the transmission of air-borne sound to the inner ear; to do so it is necessary that an osseous bridge, or connection, be established between the oval window margin and the stapedial footplate. The bridge may be narrow, but it must exist before hearing acuity is affected by an otosclerotic lesion. Rarely, if ever, is the entire annular ligament obliterated and replaced by the otosclerotic osseous tissue. I, personally, have not observed any case in which the annular ligament was not still present at some part of the circumference of the joint.

The invasion and replacement of the annular ligament usually occurs from the side of the oval window margin, but it may take place in the other direction. In those cases in which the otosclerotic process begins in the stapedial footplate, the direction of the invasion, if ankylosis occurs, must be from the footplate toward the oval window margin. This direction of spread of a diseased area is also possible, and does occur sometimes, in the formation of secondary bony bridges in ears in which an otosclerotic area primary in the otic capsule has invaded the stapedial footplate and spread through its substance.

Histological demonstration that the direction of spread of otosclerosis in an ear was really from the stapedial footplate to the oval window margin will always be difficult, in either type of case, because once the "bridgehead" in the oval window margin has been established, uncertainty exists about the direction in which the extension actually occurred. For this reason, in the report I made six years ago, only 10 ears were listed as having an otosclerotic area primary in the stapedial footplate; each of them had an area definitely limited to that structure at the time of death.[18] In the recent report by Nylén,[1c] based on his study of Nager's collection of sectioned ears, two similar cases are mentioned, and I understand that Fowler [2] and perhaps others have a few such speci-

2. Fowler, E. P., Jr.: Personal communication to the author.

mens. The fact that otosclerosis can spread from the stapes to the oval window margin, as well as in the usual direction, may be of only academic interest. I am stressing it, however, for two reasons: (*a*) because neither the possibility nor its clinical implications have been given serious consideration in the extensive literature on otosclerosis and (*b*) because the footplate of the stapes, unlike the crura or either of the two other ossicles, is, throughout development, in close contact with the primordial otic capsule. The painstaking studies of Bast and Anson[3] and their associates have conclusively settled the long discussion about the role of the otic capsule in the embryology of the stapes, and the developmental facts they have demonstrated may, if the ultimate cause of otosclerosis is ever ascertained, offer a rational explanation of the occurrence of the disease in the stapedial footplate.

Otosclerotic areas differ in size, from those less than a millimeter in diameter to those in which the disease process has involved almost the entire otic capsule. Small and moderate-sized areas occur far oftener than do the truly extensive ones.

Usually, only one otosclerotic area is present in a temporal bone, but two anatomically separate areas occur rather often, and three or more independent areas occasionally develop in an ear. For the material reported six years ago, the actual figures were as follows: 54 ears with one area each; 22 ears with two areas each, and 5 ears with three areas each. The total number of otosclerotic areas in the 81 ears was 113. Nylén[1c] reported that one of the specimens he studied had four independent otosclerotic foci.

The disease process may begin in almost any portion of the otic capsule. More than half of all otosclerotic areas arise in the region immediately in front of the oval window, and in more than three fourths of all ears with histologically demonstrable otosclerosis this region is involved. Obviously, there is good reason to call it the "area of predilection" for otosclerosis.

Anson,[4] after describing in detail the development of the region of the otic capsule immediately anterior to the oval window, expressed the opinion that the cause of otosclerosis is related to the changes that occur

3. (*a*) Bast, T. H., and Anson, B. J.: The Temporal Bone and the Ear, Springfield, Ill., Charles C Thomas, Publisher, 1949. (*b*) Cauldwell, E. W., and Anson, B. J.: Stapes, Fissula Ante Fenestram and Associated Structures in Man: III. From Embryos 6.7 to 50 mm. in Length, Arch. Otolaryng. **36**:891-925 (Dec.) 1942. (*c*) Anson, B. J., and Cauldwell, E. W.: The Developmental Anatomy of the Human Stapes, Ann. Otol., Rhin. & Laryng. **51**:881-904 (Dec.) 1942.

4. Anson, B. J.: Fissular Region of the Otic Capsule in Relation to Otosclerosis, Arch. Otolaryng., this issue, p. 843.

in the tissues of the peculiar structure known as the fissula ante fenestram. From previous friendly discussions we have had on the subject during the past twenty years or so, both Dr. Anson and Dr. Bast are well aware that on this point I do not agree with them. Two facts constitute the principal reasons for my disagreement with their otherwise plausible hypothesis about the origin of otosclerosis: (1) Otosclerosis, histologically indistinguishable from that which occurs in the area of predilection, can and often does originate in parts of the otic capsule far removed from the fissula ante fenestram, and (2) at least some of the otosclerotic areas anterior to the oval window are, when still of small size, not in contact with any portion of the fissula ante fenestram. The collection of sectioned temporal bones at Johns Hopkins contains four such specimens.[5] After an otosclerotic area anterior to the oval window has grown even to moderate size it is quite impossible to determine, from the study of histologic preparations, whether or not the area originated independently of the fissula ante fenestram, because this structure has by then been replaced by the new growth.

The round window region ranks next to the oval window in the frequency with which otosclerotic lesions develop; about one fifth of all histologically recognized areas of otosclerosis are somewhere along the zone of attachment of the round window membrane.[1a] Only rarely do areas in this region develop into major lesions, i. e., into ones that occlude the round window niche or that otherwise might affect the acuity of hearing. Likewise, the lesions in the regions of less frequent occurrence of independent development of otosclerosis seldom, if ever, grow to a size such that they, by their presence, affect the hearing. This observation is true for the independent areas in the superior, the anterior or the inferior parts of the cochlear capsule, in the part of the capsule around the semicircular canals or in the inferior part of the wall of the internal auditory canal.

Reflection on the facts that have been revealed by histological studies of otosclerosis naturally leads to curiosity about why the disease usually is innocuous. Thoughtful persons ask themselves why it is that most otosclerotic areas stop growing before they involve structures essential to the hearing function. They wonder whether the answer to this question, if known, might prove useful in the development of a rational method to stop the growth of all otosclerotic areas and thus prevent the progressive spread of those areas that cause the symptoms of clinical otosclerosis. Nutritional factors, using the term nutrition in the broadest sense, are known to be important in the control of growth in general;

5. Guild, S. R.: Early Stages of Otosclerosis, Arch. Otolaryng. **12**:457-483 (Oct.) 1930. Footnote 1a.

therefore thoughts about the growth of otosclerotic areas have naturally turned to a consideration of factors that might affect the nutritional environment of the tissues of the otic capsule.

I shall not attempt at this time to review or to summarize the studies that have been made along this line by others except to remark that thus far they have not been successful in revealing what controls either the onset or the growth and spread of otosclerotic areas. I do wish, however, to mention two morphological facts that are of interest and that should be given consideration in thinking about the possible role of nutritional factors in otosclerosis. The first fact is that otosclerosis occurs unilaterally. In my studies, about 30 per cent of the persons with histological otosclerosis had only one ear involved. Nylén found a similar proportion, "somewhat over 25 per cent," of unilateral otosclerosis in the material he studied. The second fact, which is the more important one from the standpoint of the growth as distinct from the pathogenesis of otosclerotic areas, is that in persons with bilateral otosclerosis, bilateral symmetry of the areas is not always present. Not only may the otosclerotic areas in the two ears of a person differ in location and extent, but similarly located areas may differ from each other greatly with respect to the stage of activity present. The histological appearance of the sectioned area in one ear may, for instance, afford good evidence that the lesion was growing actively during the period immediately preceding death, while the appearance of the otosclerotic area in the other ear is that of complete quiescence for a long time. Attempts to explain the pathogenesis of otosclerosis and the growth of otosclerotic areas after their inception should take into account the above morphological facts, unilateral occurrence of the disease and bilateral differences in growth activity of diseased areas, together with the physiological fact that, in any person, the blood has supplied both otic capsules with the same nutritional factors, for identical lengths of time, irrespective of temporary or permanent deviations of any of the factors from the range of normal.

Despite all that has been learned about otosclerosis, its basic riddle remains unsolved.

OTOSCLEROSIS

Hypothesis of Its Origin and Progress

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THE DIFFICULTY in giving a precise characterization of the histopathology of otosclerosis has long been recognized. Yet the microscopic picture is clearcut and easily identified. How can such a paradox be?

A wealth of clinical and microscopic knowledge that comprises a multiple picture of otosclerosis has been inherited from men of great integrity. What is the significance of this fact?

The architectonic structure of haversian systems in the normal otic capsule is very complicated. Because of the irregular shape of the capsule, the blood vessels supplying it are forced to make rapid and complicated curves in their course through it. Consequently, in every microscopic section there is a variety of patterns of bony lamellas. Very occasionally, haversian canals will be cut at right angles to the long axis of the vessel, thereby giving the textbook picture of a haversian system. Often, they will be cut at various tangents, and will give, accordingly, an intricate and irregular pattern of lamellas. In certain areas the lamellas, and even the vessels themselves, will be cut in a longitudinal plane (fig. 1).

With the intricacies of the normal anatomy in mind, I shall discuss the pathological picture when otosclerosis is present.

The otosclerotic lesion presents a circumscribed area of spongy pathological bone clearly demarcated from the normal bone. The contour of the diseased area is usually more or less scalloped, following the pattern of haversian systems. The disease is definitely seen to advance

From the Lempert Institute of Otology.

This paper is the third in a "Symposium on Histology and Pathogenesis of Otosclerosis," held at the meeting of the Otosclerosis Study Group, Division of the Committee on Conservation of Hearing, in connection with the Fifty-Fourth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Chicago, Oct. 8, 1949.

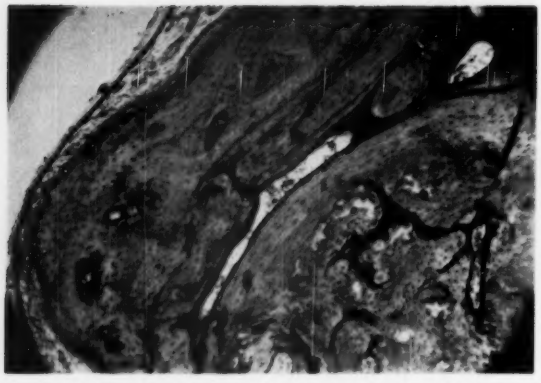

Fig. 1.—The intricate and irregular architectonic structure of haversian systems in the normal otic capsule of the adult. Interspersed among the globuli interossei are blue mantles. Lamellas may appear in concentric formation or in linear formation, according to the angle of cutting.

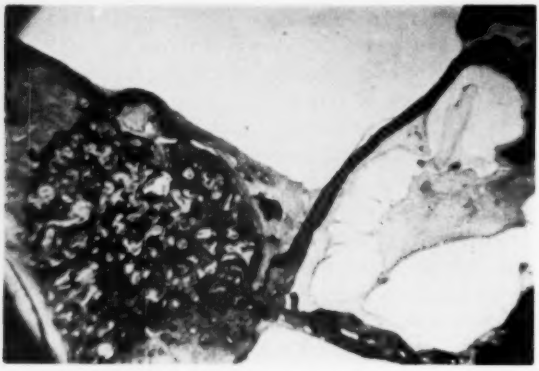

Fig. 2.—A typical case of otosclerosis. The lesion lies anterior to the footplate of the stapes. Ankylosis has occurred. The footplate is also involved. The pathologic bone is pitted with vascular and fibrotic spaces. The contour of the lesion presents a scalloped appearance because the disease advances by haversian systems. Attack on the next haversian canal is evident in the upper left field. The otosclerosis was bilateral. Washington University Collection; courtesy of Williams & Wilkins Company.

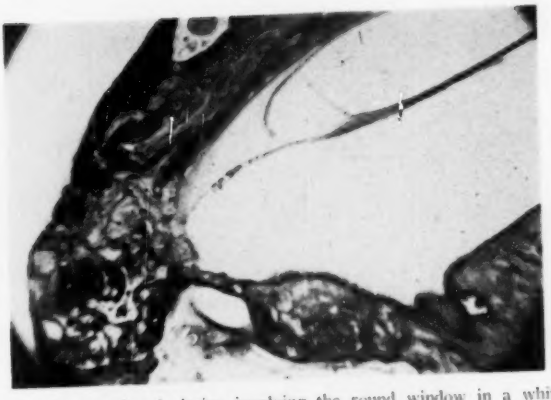

Fig. 3.—An otosclerotic lesion involving the round window in a white man aged 21. The oval window was not involved. A fracture line occurred between the niche of the round window and the ampulla of the posterior canal, as shown in figure 11. Washington University Collection, 7010.

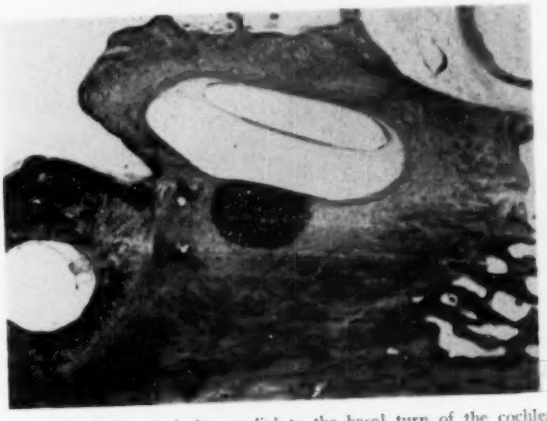

Fig. 4.—An otosclerotic lesion medial to the basal turn of the cochlea, near neither window, in a white man aged 46. In this location no known clinical symptoms can be elicited. Washington University Collection, 8630.

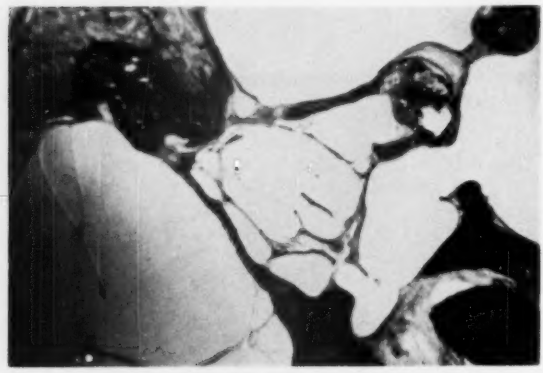

Fig. 5.—Otosclerosis may involve the region of the fissula ante fenestram. Washington University Collection, 5178.

Fig. 6.—A diffuse type of otosclerosis involving the inferior border of the facial canal, the oval window, promontory, round window and the border of the posterior ampulla (Dr. Lempert's case).

along blood channels. Congestion and stasis are a frequent observation, as Wittmaack [1] noted.

The common site of occurrence is in the oval window (fig. 2), but the condition may occur also in the round window (fig. 3); between the two windows (fig. 4); in the internal auditory meatus; near the apex of the cochlea; near the superior semicircular canal, or in the external auditory canal. The lesion may involve the region of the fissula ante fenestram, as discussed by Bast, Anson and Guggenheim (fig. 5). Although usually circumscribed, it may be diffuse (figs. 6, 7 and 8).

The blood vessels in cases of otosclerosis seen under the microscope exhibit blue mantles. They were described by Manasse.[2] Manasse [2]

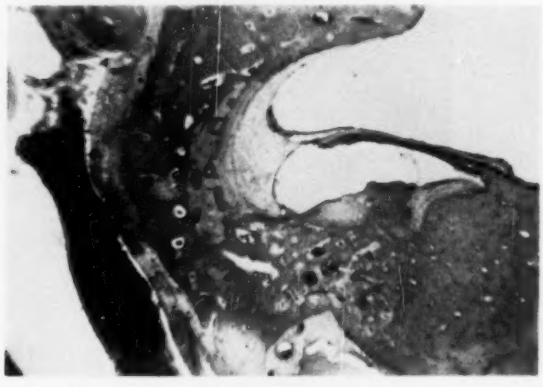

Fig. 7.—Involvement of the promontory and the niche of the round window in a white woman aged 47. Note obliteration of the membrana tympani secundaria, with protrusion of atypical bone into the scala tympani, and that the attack on the inner bony promontory wall is around blood vessels (Dr. Lempert's case 1).

and Nager [3] recognized that resorption of bone along these blue mantles could occur without the aid of osteoclasts. Nylén,[4] on the other hand,

1. Wittmaack, K.: Die Ursache der Otosklerose: Ein Vorschlag zur ursächlichen Behandlung, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **129**:150-173 (June 26) 1931.

2. Manasse, P.: Neue Untersuchungen zur Otosklerosenfrage, Ztschr. f. Ohrenh. **82**:76-96, 1922.

3. Nager, F. R.: Zur Klinik und pathologischen Anatomie der Otosklerose, Acta oto-laryng. **27**:542-551, 1939.

4. Nylén, B.: Histopathological Investigations on the Localization, Number, Activity and Extent of Otosclerotic Foci, Upsala läkaref. förh. **54**:1-52 (March 15) 1949.

Fig. 8.—Osteoporotic changes seen in the upper field. Hypervascularity, congestion, stasis and fibrosis appear in the central field. Inferiorly, the pathologic extension occurs along vascular channels (Dr. Lempert's case 1).

Fig. 9.—Microscopic evidence of extension of pathologic chemical fluid from blue-mantled haversian canals through canaliculi to adjacent lacunas, having the appearance of insects walking along a stick. The neighboring lacunas are devoid of bone cells, thereby indicating necrosis (detail from fig. 8).

Fig. 10.—Longitudinal section of a haversian canal passing through otosclerotic bone. Note the shadowy braided structure at an expanded region just below the center of the field, the "breccia," or braided bone. Note also the endothelial cell with crenulated cytoplasm in the lower portion of the field.

Fig. 11.—The otosclerotic lesion may not occur at the site of the fracture line. The fracture line occurs because of the loss of support induced by the otosclerotic process. Washington University Collection, 7010.

writing in 1949, does not believe that blue mantles are significant because "they occur around semicircular canals where the disease is rarely seen."

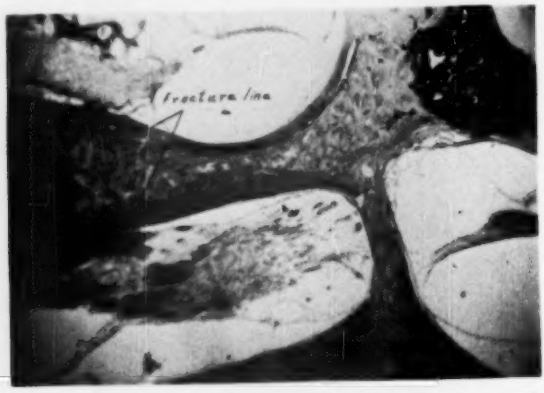

Fig. 12.—Fracture lines occurring because the petrosa has been weakened by the decalcification of the bone. The petrosa is the support for the weight of the brain and the dome of the skull. Fractures will occur wherever the strain is too great for the remaining bone to bear, not necessarily in the area of otosclerosis.

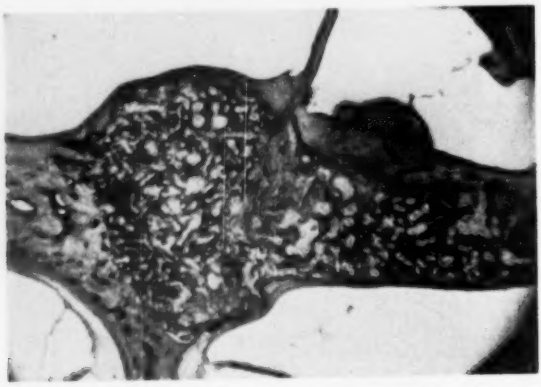

Fig. 13.—The thickened footplate of the stapes, covered by thickened mucosa containing congested vessels in a white woman aged 23. Extensive ankylosis has occurred in this case with a history of deafness for nine years (Dr. Lempert's case 2).

I believe that blue mantles may be considered as chemical indicators, so to speak, of the type of calcium being deposited or existing along the course of a vessel. Blue mantles are observed in temporal bones that

are not otosclerotic, but they are a prominent characteristic of otosclerosis. The spread of the pathological fluid from the blood stream can be traced through the canaliculi to the adjacent lacunas. The lacunas look like insects walking along a stick (fig. 9). At a later stage, the presence of the fluid can be traced along the canaliculi on the other side of these lacunas to those not in immediate contact with a haversian canal. Osteoclasts may be present in such great numbers that osteoclasia has been considered as a diagnosis. In some specimens no osteoclasts have been seen.

Weber[5] and others stressed the significance of the braided bone that appears in otosclerosis, the so-called breccia. Figure 10 illustrates a haversian canal in an otosclerotic center of bone. The somewhat

Fig. 14.—The oval window shown at a lower level than in the preceding illustration. Note the large tumor-like mass of bone lying between the crura of the stapes. Its origin may be seen in figure 15. The round window was normal.

shriveled endothelial cells on the wall of the vessel can be seen. This condition of the cells allows the detrimental fluid to pass through. Evidence of a widening or decalcification of the bony canal is seen below center. Here, also, are noted parallel bands interlacing each other. At this stage they are mere shadows of old cement lines. At a later stage, when repair has set in, they will present the picture illustrated in figure 17 of the report on ossicles in otosclerosis by Lempert and Wolff.[6] In

5. Weber, M. H.: The Blue Mantles in Otosclerosis: A Contribution to the Pathology of the Labyrinthine Capsule, Ann. Otol., Rhin. & Laryng. **42**:438-454 (June) 1933.

6. Lempert, J., and Wolff, D.: Otosclerosis: Theory of Its Origin and Development, Arch. Otolaryng. **50**:115-155 (Aug.) 1949.

this illustration are seen dense fiber bone with loss of normal haversian system contours and an enlargement of lacunas, which show up very well in unstained sections when examined in polarized light, as was done by Weber.

Sometimes the disease definitely assumes the characteristics of a tumor of the bone. Mayer,[7] observing this fact, concluded that it was a vascular tumor. He called it a hemartoma and thought that it arose from nature's attempt to heal a fracture of the otic capsule. To him, it was something like a callus. In the small collection of cases at the Lempert Institute are three examples of fracture lines. None of these occur through the pathological bone, thereby forcing one to conclude that the fractures occur because of the disease. The spongification of

Fig. 15.—The origin of the hemangiomatous tumor, the promontory wall. Congestion and stasis of vessels, fibrosis of the bone and osteoclasia were all present.

the bone has altered the normal architectural bulwark of the petrosa as a support to the dome of the skull and the weight of the brain. Hence fractures occur (figs. 11 and 12).

There are times when the disease assumes the characteristics of a hemangioma of the bone, as is evident in the case illustrated in figures 13, 14 and 15. Similar small tumors of bone have been found elsewhere in the skeletal anatomy. Horwitz [8] found one in a digit. Jaffe [9] reported the presence of osteoid osteomas in the long bones. Studying his

7. Mayer, O.: Die Ursache der Knochenneubildung bei der Otosklerose, Acta oto-laryng. **15**:35-73, 1931.

8. Horwitz, T.: Localized Aseptic Necrosis and Rarefaction of Bone, Radiology **38**:364-367 (March) 1942.

9. Jaffe, H. L.: Osteoid-Osteoma of Bone, Radiology **45**:319-334 (Oct.) 1945.

specimens and those of my associates and mine in his laboratory, both of us were impressed with the similarity in some of the microscopic pictures. The tumors in Jaffe's cases produced symptoms of pain. Pain has not been recorded as a symptom in otosclerosis. How often "headache" appears in the history, the clinician must know. Kelemen [10]

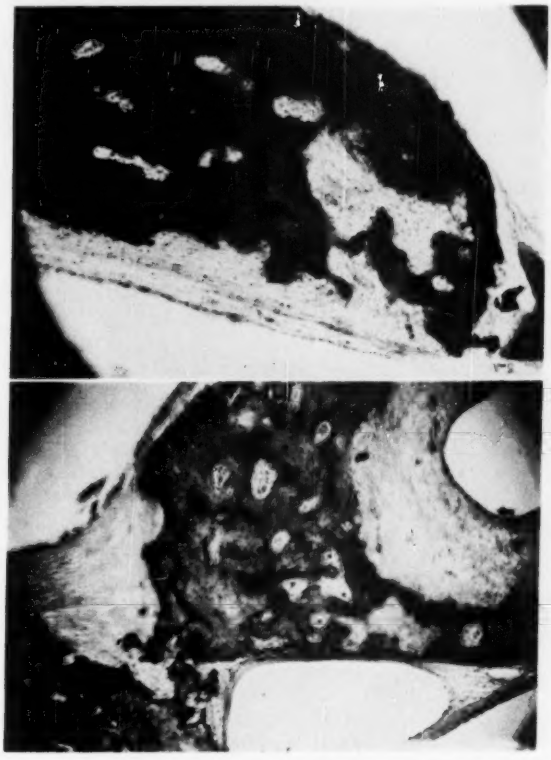

Fig. 16.—Osteofibrosis and overgrowth of pathologic bone in the footplates of the right and left stapes in a case of bilateral otosclerosis (courtesy of Dr. Kinney).

found an osteoma in the frontal sinus which "simulated the final stage of an otosclerotic lesion." Similar osteomas have been reported in the external auditory canals of otosclerotic patients.

10. Kelemen, G.: Otosclerotic Focus Outside the Inner Ear Capsule, Laryngoscope **53**:528-532 (Aug.) 1943: Frontal Sinus Osteomata, Acta oto-laryng. **27**:528-541, 1939.

Nager and Meyer [11] at one time thought of otosclerosis as a localized form of osteitis deformans (Paget's disease). Osteitis fibrosa cystica is now recognized as nature's reaction to rapid decalcification of bone, illustrated in figure 16. When ankylosis of the stapedial footplate occurs in such cases, a diagnosis of otosclerosis will be made.

Gerlings and de Kleijn [12] found otosclerosis in a case of gargoylism. In a case of gargoylism (dysostosis multiplex) I found petrosae in which there was no evidence of otosclerosis, but there were joint changes in the ossicles and microscopic porosity of the petrosa as a whole.[12a]

Otosclerosis can also be shown to be a localized form of osteoporosis, illustrated in figures 4 and 17. Evidently after excessive vascularization

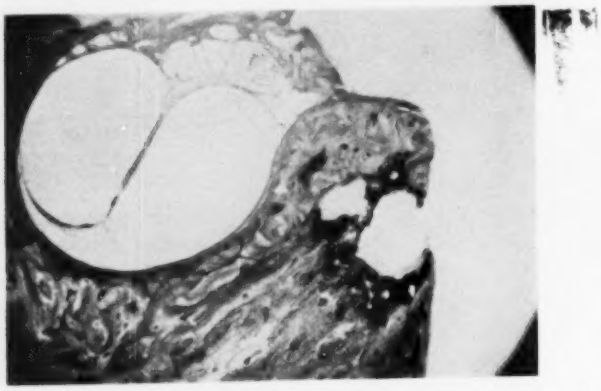

Fig. 17.—Complete dissolution of bone in the internal auditory meatus in the same case in which the isolated otosclerotic area of osteoporotic type was seen along the base of the cochlea. Note the atrophy of the stria vascularis, the organ of Corti and the spiral ganglion. Washington University Collection, 8630.

had occurred, an embolus or obliterative arteritis blocked the blood supply to the area and produced the pathologic changes observed. It is interesting to note the lesion in the internal auditory meatus where the tissue fluids have simply washed the necrotic bone away without any reaction toward repair (fig. 17).

11. Nager, F. R., and Meyer, M.: Die Erkrankungen des Knochensystems und ihre Erscheinungen an der Innenohrkapsel des Menschen, Berlin, S. Karger, 1932.

12. Gerlings, P. G., and de Kleijn, A.: Some Remarks About Gargoylism, Proc. koninkl. nederl. Akad., 1946.

12a. Wolff, D.: Microscopic Study of Temporal Bones in Dysostosis Multiplex (Gargoylism), Laryngoscope **52**:218-223 (March) 1942.

Otosclerosis may be accompanied with increased pressure within the labyrinth, as was pointed out by Meyer.[13] Figures 18, 19 and 20 illustrate a case of Ménière's syndrome. The diagnosis was not clearcut. As can be noted, otosclerosis is present, but no clinical diagnosis was made. There is no ankylosis of the stapes. Hydrops of the labyrinth is present in both ears. In addition, in the vestibular ganglion of the right ear is an area of atrophy of ganglion cells. This atrophy was found to be due to obliterative arteritis. In its end stage, one cannot determine whether the lesion developed from obliterative periarteritis, as in periarteritis nodosa, or from obliterative endarteritis. The patient was a dentist and suffered from severe disorientation and dizziness. It is not impossible that similar lesions may also have occurred intra-

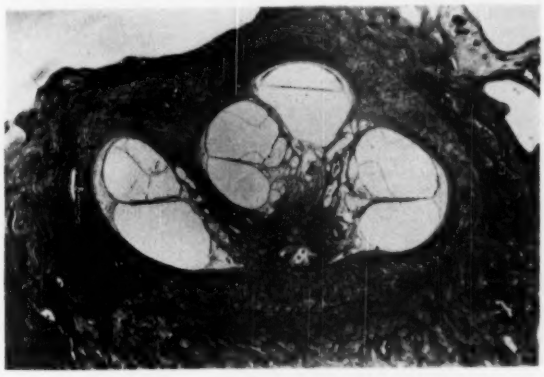

Fig. 18.—A typical picture of a distorted Reissner's membrane in hydrops of the labyrinth in a white man. Washington University Collection, 5178 left.; 1052.

cranially to obscure diagnosis further. Druss and Maybaum [14] reported two cases of periarteritis nodosa of the temporal bone with otosclerosis. The diagnosis was generalized periarteritis nodosa.

It is well known that otosclerosis may be found in cases of blue sclera.

Thus it is evident that otosclerosis may assume many forms. It has multiple etiologic factors. It may occur in any case with bone or calcium dyscrasia. It may arise in any case in which peripheral vascular

13. Meyer, M.: Eine neue Anschauung über die Schwerhörigkeit bei Otosklerose und vielleicht ein neuer Weg zu ihrer Behandlung! Acta oto-laryng. **27**:1-15, 1939; Monatsschr. f. Ohrenh. **73**:140-152 (Feb.) 1939.

14. Druss, J. G., and Maybaum, J. L.: Periarteritis Nodosa of the Temporal Bone, Arch. Otolaryng. **19**:502-507 (April) 1934.

pathologic conditions exist. These changes may block the normal release and the resorption of calcium in an area.

A peripheral vascular pathologic condition arising from any cause explains the bilateral symmetry of the disease. It is well known that

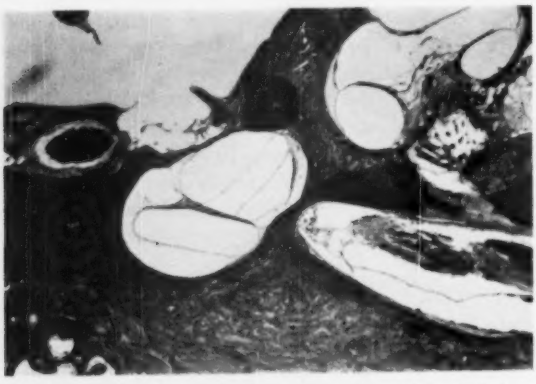

Fig. 19.—Distortion of the membrane in the cochlea and vestibule in a case of bilateral hydrops of the labyrinth and otosclerosis. Washington University Collection, 5178 right.

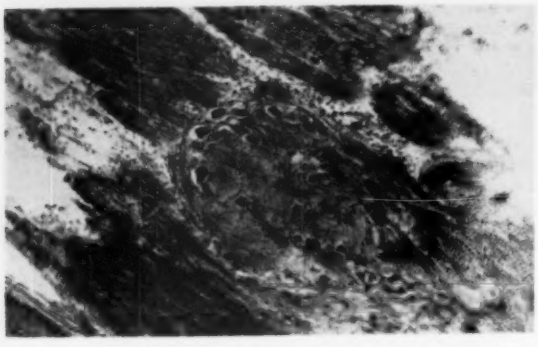

Fig. 20.—Obliterative endarteritis in the artery supplying the vestibular ganglion. This lesion produced extensive atrophy of the ganglion, with clinical symptoms. Washington University Collection, 5178 right.

the same phalanx of the same digit in bilateral extremities may simultaneously be affected in this disease.

Peripheral vascular disease accounts for the decalcification process. Sluggish blood in excess numbers of capillary loops has been observed

in the nail beds of patients with this condition. Decalcification of bone commonly occurs in the presence of stasis.

Peripheral vascular disease explains why in some cases there is atrophy of the stria vascularis, the spiral ganglion and the organ of Corti. It means that the internal auditory artery is also affected. Hydrops of the labyrinth is explained in the same way. This disease explains why the loss of word hearing is sometimes greater than the audiometer record for pure tones indicates that it should be.

Tinnitus may possibly be due to alterations in vascular channels such as the inferior tympanic artery or, indeed, others in the area.

When ankylosis of the footplate, immobilization of the other two ossicles or atrophy of the muscles in the middle ear due to loss of blood supply occurs, one can imagine that the clinician will note these changes reflected in the audiometer record. Whether there are two types of records for involvement of the two windows, I do not know.

I predict that when a complete reconstruction of the detailed terminal blood supply of the adult petrosa is made, one will find the areas of more or less terminal capillaries with little or no chance for anastomoses to be the sites of predilection for otosclerosis.

The incipient and early progressive stages of the disease and the various microscopic aspects it may assume have recently been reported in the literature by Lempert and Wolff.[6] Study of his large collection of ossicles has led us to the hypothesis that otosclerosis has no specific cause but that it is the result of damage to the peripheral blood supply. This damage might be chemical, vasomotor or allergic. It may be trauma from vibratory phenomena. What trend the ultimate course of the disease will take depends on the constitution of the individual patient. In some patients there may be a hemangioma; in others, fibrosis will occur, and in still others osteoporosis may develop.

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INFLUENCE OF SYSTEMIC AND LOCAL FACTORS ON THE DEVELOPMENT OF OTOSCLEROSIS

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INVESTIGATIONS which have been made in the search for evidence of a relation between otosclerosis and other systemic diseases have so far yielded little positive information as to the etiology of otosclerosis. Attention has been directed to various diseases of glands of internal secretion, hormone imbalance, vitamin deficiencies and metabolic disturbances, but laboratory investigations have so far revealed no specific connection.

The presence of a hereditary predisposition to otosclerosis has been demonstrated in as high as 50 per cent of some large series of clinical cases. Although the disease is known to exist without clinical symptoms in a large percentage of people and the application of mendelian laws is therefore impractical, the existence of a hereditary factor seems definite. Clinical observation has provided some evidence that certain constitutional factors may exert some influence on the progress of the disease, perhaps also on its development. The possibility exists that certain constitutional states may furnish the environmental influence necessary to activate the disease.

Histopathological studies have so far given basic information about the development and structure of the labyrinthine capsule and the changes which occur in it as a part of the various diseases affecting bone. These studies have added greatly to the understanding of the otosclerosis problem, but more definite information as to etiological factors has been lacking.

A review of the available information regarding constitutional and local factors which may influence the disease is in order in the search for possible leads for further investigation.

From the Division of Otolaryngology, University of Chicago.

This paper is the fourth in a "Symposium on Histology and Pathogenesis of Otosclerosis," held at the meeting of the Otosclerosis Study Group, Division of the Committee on Conservation of Hearing, in connection with the Fifty-Fourth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Chicago, Oct. 8, 1949.

The special structure of the labyrinth capsule is well known, and the development of the three layers has been accurately followed.[1] The lack of necessity for further growth of the labyrinthine spaces after fetal life has made possible the peculiar structure of the middle, or endochondral, layer of the capsule, in which the primary bone with its enclosed spicules of calcified cartilage persists throughout life.

The endosteal and endochondral, or enchondral, layers have an unusually dense character and show a special resistance to most disease processes which readily affect the outer periosteal layer. The endochondral layer is also known to exhibit comparatively little osteogenetic activity following fracture. Healing of the fracture takes place mainly from the endosteal and periosteal surfaces.

The periosteal layer of bone continues to be added to during growth of the base of the skull, thereby increasing the over-all dimensions of the pyramid. Marrow spaces may persist in this layer in areas surrounding the capsule as well as in the apical part of the pyramid. Pneumatization also is limited to the periosteal layer.

EFFECT OF VARIOUS DISEASES OF THE BONY SKELETON ON THE LABYRINTHINE CAPSULE

There are a number of extensive reports on the histopathological changes in the petrous pyramid in the presence of various diseases affecting bone. Investigations by Siebenmann, Manasse, Wittmaack, Nager, Mayer, Meyer, Brunner, Weber, Wilson and Anson and many others have shown the extent to which the different layers of the capsule tend to be affected by diseases affecting the skeleton as a whole. These reports are included in the résumé of the literature by the Central Bureau of Research of the American Otological Society, Inc., in four volumes, entitled "Otosclerosis."[2]

Ricketts has been shown to affect mainly periosteal bone of the pyramid and to have no relation to otosclerosis. The changes in senile osteoporosis and osteomalacia have been mainly in periosteal bone. The endochondral layer has shown little evidence of absorption. The endemic deafness of the Alpine regions, associated with cretinism, bone deformities and dwarfism, is related to the deposition of new periosteal

1. Bast, T. H., and Ason, B. J.: The Temporal Bone and the Ear, Springfield, Ill., Charles C Thomas, Publisher, 1949.

2. Duel, A. B.: Otosclerosis: A Résumé of the Literature to July 1928, volumes 1 and 2, American Otological Society, Inc., New York, Paul B. Hoeber, Inc., 1929. Duel, A. B., and Fowler, E. P.: Otosclerosis: A Résumé of the Literature, 1928-1935, volume 3, American Otological Society, Inc., St. Louis, Annals Publishing Company, 1936. Fowler, E. P.: Otosclerosis: An Index of the Literature with Abstracts and an Index of Authors According to Subjects, 1936-1942, volume 4, New Bedford, Mass., American Otological Society, Inc., 1943.

bone to the promontory with narrowing of the window niches, interference with, and sometimes malformations of, the ossicles.

Osteopetrosis (Albers-Schönberg's disease, marble bones or osteosclerosis fragilis) has been characterized by a deposition of enormous amounts of dense periosteal bone. The labyrinthine spaces have been found free from encroachment and the sensory structures not involved. The deposition of an extreme thickening of new periosteal bone has resulted in extensive changes affecting middle ear spaces and the course of nerves through the periosteal layer, but the endochondral and endosteal layers are clearly recognizable and have not been replaced.

Dyschondroplasia (chondrodystrophy) has not been associated with any apparent tendency toward otosclerosis in the case of those who have reached adult life. The same alterations in the ossification zones of the pyramid have been observed as are known to occur in extremities, leading to dwarfism. In osteitis deformans, or Paget's disease, the endochondral layer does not escape the disease process. In those cases in which the temporal bone is involved the destruction and new formation of bone start from the periosteal layer but involve all layers eventually. The type of deafness may be conduction in early stages, due to impedance at the stapedeovestibular joint, but a progressive nerve deafness is characteristic. Sometimes a sudden episode of deafness and dizziness has occurred in the late stages.

The histopathological alterations in osteitis deformans are similar in detail to those seen in some areas of active otosclerosis. The distribution and rate of progress, however, are quite different. The cause of osteitis deformans is also unknown, and nothing has been found as yet that will appreciably influence the progress of the disease.

The type of osteodystrophy seen in generalized osteitis fibrosa cystica of Recklinghausen, with tumors of the parathyroid gland, has shown resemblance to some areas of an active otosclerotic nodule. Nager and Meyer and Weber considered otosclerosis to be a local osteodystrophy.

The frequent association of blue scleras, fragile bones and otosclerosis and the hereditary tendency of this syndrome (van der Hoeve-de Kleijn syndrome) have been of especial interest. The familial tendency has been studied in many family trees for three and four generations. According to Fowler,[4] the incidence of blue scleras in patients with otosclerosis was found to be 69 per cent and the incidence of otosclerosis in those with marked blueness of the scleras, 50 per cent. The incidence of otosclerosis in persons with osteogenesis imperfecta cannot be

3. Footnote deleted.

4. Fowler, E. P.: The Incidence (and Degrees) of Blue Sclera in Otosclerosis and Other Disorders, Laryngoscope **59**:406-416 (April) 1949.

estimated, since the deafness appears only in the second or third decades whereas the majority of children born with osteogenesis imperfecta do not survive to adult age.

Not all children with blue scleras in affected families have fragile bones, but in such families the existence of the triad in those who have reached adult life is frequent. Examination has shown that mainly the periosteal layer of the capsule has been affected. In the performance of the fenestration operation on such a patient the bony trabeculae were found to have a strikingly fine and delicate character, with the bony labyrinthine capsule of apparently normal hardness but thinner than usual.

The occurrence of this syndrome has been of especial interest from the standpoint of otosclerosis. However, little is known about the origin of osteogenesis imperfecta or of blue scleras. The nature of the relation to otosclerosis is not definite, although both diseases have shown a hereditary tendency.

REACTION OF THE LABYRINTHINE CAPSULE TO LOCAL FACTORS SUCH AS INFECTION AND TUMOR

Inflammatory disease which enters the pyramid by way of the air spaces affects mainly the periosteal bone. The endochondral and endosteal layers offer great resistance to invasion, but in the event of the process becoming walled off in a group of air cells or in marrow spaces the endochondral layer, as well as the endosteal layer, may eventually break down. Direct resorption of all layers may then occur along the surface and along vascular spaces, or sequestrums may form, presumably as a result of obliteration of blood supply.

A suppurative process within the labyrinth may invade all layers of the bony capsule. The greater density of the endosteal and endochondral layers, however, provides for greater resistance to invasion by infection on the part of these layers than in the case of the periosteal layer. The latter owes its vulnerability probably to the presence of air spaces and vascular and marrow spaces.

Similarly, in the presence of tumor the endochondral and endosteal layers resist invasion and absorption more effectively than does the periosteal layer. This may be accounted for, in part at least, by the absence of air and marrow spaces and the relative avascularity of the middle and inner layers of the capsule.

CONSTITUTIONAL FACTORS

There has been no conclusive evidence from clinical observation that disturbances of internal secretion or hormones or vitamin imbalance have been related to otosclerosis. The clinical impression exists, however that pregnancy has some tendency to activate the disease and also

that prolonged illness may have a similar influence. The statistics presented by Guild [5] appear to indicate a higher frequency of otosclerosis in persons who have died between the ages of 30 and 49 than in persons in the corresponding period over the age of 49. The significance is not readily apparent but the question arises as to whether the incidence in normal healthy people in this age period would be similar to that in the older age group.

The incidence of otosclerosis found at autopsy may depend in part on the material available; for example, the type of institution, the predominant age group, the incidence of intracranial disease and the special interests of the collector. Large collections from normal healthy adults may show a lower incidence. Available reports seem to indicate the approximate incidence of around 10 per cent in material from white adults examined at autopsy.

From my collection of *22* bones showing definite otosclerosis, the question has arisen as to whether some of the histological nodules found could have originated during the terminal illness and, if so, whether that illness was a necessary environmental factor in the etiology of otosclerosis.

The collection contains four bones which were obtained because the person was known to have otosclerosis; otherwise all cases were found only on routine postmortem examinations. The majority of these came, however, from patients with intracranial disease, such as tumor, osteomyelitis and meningitis.

Specimens from three cases (six ears) show what may be described as a very early active stage of the disease. These cases have raised the question as to whether the prolonged terminal illness may have been the environmental factor which activated the otosclerosis in a person with a hereditary tendency.

REPORT OF CASES

CASE 1.—E. A., a girl aged 9 years who died from intracranial malignant neoplasm which was known to have existed for about one year, was examined at autopsy. Examination of the temporal bones showed essentially normal middle and inner ears, but in the labyrinth capsule, just in front of the oval window, on each side was a small well demarcated area of pathological bone which exhibited a very early active stage of disease (fig. 1). In some areas osteoclasts were visible bordering both on the otosclerotic bone and on the primary bone of the endochondral layer; rows of osteoblasts were also visible in some of the enlarged marrow spaces. Vessels were prominent in this fibrotic type of marrow. In one ear extension could be observed along a vascular space at the margin of the otosclerotic nodule (fig. 2). The nodules were similar in size and in apparent activity. The periosteal and endochondral layers were both involved. The calcified cartilage along the oval window and fissula did not appear to be primarily affected, although involved to some extent.

5. Guild, S. R.: Histologic Otosclerosis, Ann. Otol., Rhin. & Laryng. **53**:246-266 (June) 1944.

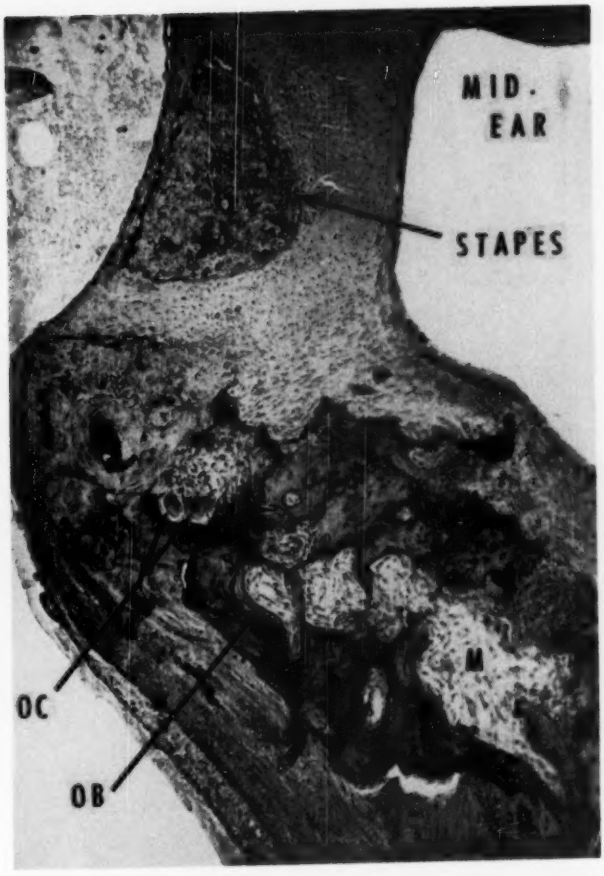

Fig. 1 (case 1).—Section of ear through lower anterior margin of oval window and stapes footplate of a girl aged 9 years. A clearly demarcated area of pathological bone is shown which involves periosteal and endochondral layers of the capsule. Fibrotic marrow spaces (*M*), osteoclastic resorption of both otosclerotic bone and normal bone of the capsule (*O.C.*) and laying down of new bone by osteoblasts (*O.B.*) are shown.

Fig. 2.—Same ear as figure 1, section immediately anterior to the oval window, showing calcified cartilage at the margin of the oval window (*C. C.*), and fibrous tissue in the submucosa (*M.*). Extension of the otosclerotic focus (*F.*) along a vascular space is indicated by the lower arrow.

The histological diagnosis was bilateral otosclerosis in an early and active stage.

CASE 2.—L. J., a woman aged 19 who died from meningeal sarcoma which had invaded one internal auditory meatus, was examined at autopsy. The middle ears, as well as the labyrinthine spaces, were essentially normal, although the tumor tissue extended in to the limits of the meninges on one side (fig. 3).

Fig. 3 (case 2).—From a woman aged 19, with meningeal sarcoma and bilateral otosclerosis. Horizontal section through stapes footplate, in front of which is seen a small focus of otosclerosis (*O.F.*) in which are many dilated blood vessels (*B.V.*). Tumor tissue has extended into the internal meatus to the limits of the meninges.

In the area of predilection in front of the oval window a relatively small focus of otosclerosis was present with signs of active bone destruction, many marrow

spaces containing fibrous marrow and dilated blood vessels. Extension along vascular spaces was evident in several areas in each focus. All layers of the capsule were involved, and there was considerable thickening of the annular ligament but no absorption of the calcified cartilage which forms the margin of the stapes footplate.

Fig. 4 (case 3).—From the right ear of a man aged 30, with scarlet fever, osteomyelitis of the sphenoid bone and meningitis. Bilateral small focus of otosclerosis at the attachment of the round window membrane (*R. W. M.*).

The histological diagnosis was bilateral otosclerosis in an early and active stage.

Case 3.—T. K., a man aged 30 who died of meningitis resulting from osteomyelitis of the sphenoid bone complicating scarlet fever, was examined at autopsy. Both temporal bones showed evidence of inflammatory reaction in the middle ears

without frank suppuration and an early stage of extension of the meningitis to the labyrinth.

The labyrinthine capsule was normal in all areas except at the outer margin of the attachment of the round window membrane to the bone. Here a very small

Fig. 5 (case 3).—Left ear; high power magnification of small focus of otosclerosis at the attachment of the round window membrane showing dilated vessels (*B.V.*), osteoclastic resorption (*O.C.*) and new bone formation by osteoblasts (*O.B.*).

localized area was present bilaterally showing active resorption of bone, fibrotic and highly vascular bone marrow and laying down of new bone (figs. 4 and 5).

The size and activity of the areas were such as to indicate a probable recent beginning of the process.

The histological diagnosis was bilateral otosclerosis, in an early stage.

Comment.—These three cases present the early stage of otosclerosis. In four ears the disease was located at the area of predilection in front of the oval window and in two ears at the next commonest site, the attachment of the round window membrane at its lateral margin.

Fig. 6 (case 4).—From a youth aged 19, with pernicious anemia and unilateral healed chronic inflammatory middle ear disease with associated otosclerosis. Horizontal section at the level of the stapes footplate showing retracted and distorted tympanic membrane (*T.M.*) with defect posteriorly, fibrosis in the middle ear cavity (*Fib.*), otosclerotic focus (*O.F.*), eustachian tube (*E.T.*) and carotid canal (*C.C.*).

The stage of activity evident on histological examination and the minute size of the nodules indicate that they might well have originated within a period of weeks and thus may have represented the effect of a prolonged terminal illness on a subject already possessing the hereditary tendency.

Case 4.—S., a youth aged 19 who died of pernicious anemia, was examined at autopsy. The left temporal bone was normal. Pneumatization was extensive, and

the inner and middle ears and the bony capsule were normal. The right temporal bone showed almost complete absence of pneumatization of the mastoid process with a distorted and damaged tympanic membrane, adherent to the medial wall of the middle ear, the result of former suppurative disease which had healed (fig. 6). The inner ear was normal. The labyrinthine capsule of this ear contained a well demarcated nodule of otosclerosis in the area in front of the stapes. Signs of activity were much less than in the cases described above, and sclerosis was more advanced. There was no evidence of involvement of the stapes.

Comment.—The association of otosclerosis in one ear with healed chronic inflammatory disease and absence of pneumatization, while the opposite ear was completely normal and well pneumatized, supports the opinion expressed by some former observers that a local pathological process such as chronic inflammation might provide the necessary factor for activation of otosclerosis in the presence of a hereditary disposition.

These four cases seem to give evidence to support the belief that some environmental factor, either through a local effect or a constitutional influence, may play an important part in starting the disease process in a subject already possessing a hereditary tendency.

EXPERIMENTAL EVIDENCE

Otosclerosis has never been observed in lower animals. Experimental efforts to produce a lesion resembling the disease have so far failed. The greatest resemblance to the human labyrinthine capsule is found in monkeys, in which the endochondral layer contains cartilaginous remnants, and even some evidence of the fissula antefenestram may be found.

Experiments reported in the literature have included the effect of dietary restrictions and excessive use of certain hormones and certain drugs and chemicals.

Young animals exposed to a diet deficient in vitamin A [6] have shown extensive development of new bone, which was superimposed mainly on dural surfaces of the skull, resulting in constriction of nerve channels and pinching of nerves. The endochondral and endosteal layers of the capsule showed no significant changes, probably because growth of the labyrinthine spaces had been completed.

Injection of large doses of parathyroid extract (para-thor-mone®) has produced a type of osteitis fibrosa in young dogs and has been

6. Mellanby, E.: Croonian Lecture: Nutrition in Relation to Bone Growth and the Nervous System, Proc. Roy. Soc., London s.B **132**:28-46 (March 1) 1944. Perlman, H. B.: Effect on the Ear of Vitamin A Feeding After Severe Depletion, Arch. Otolaryng. **50**:20-35 (July) 1949. Wolbach, S. B., and Bessey, D. A.: Vitamin A Deficiency and the Nervous System, Arch. Path. **32**:689-722 (Nov.) 1941.

reported to cause changes in the labyrinthine capsule similar to otosclerosis.[7]

In 1932 a series of four monkeys were given injections of parathyroid extract in large doses for a period of four months,[8] but the injection failed to produce any definite change in the labyrinthine capsule.

I have observed electrocoagulation of the labyrinthine capsule in monkey experiments, to be followed by active resorption of the affected bone and replacement by new bone. The endochondral layer is affected as actively as the periosteal layer. The resportion takes place along vascular channels. These become dilated as bone is absorbed from the walls and filled with fibrous marrow. As the old bone is removed new bone is eventually deposited to fill the defect.

SUMMARY

The attempt to link up otosclerosis with other general constitutional factors has so far demonstrated no definite relation either on clinical or on experimental investigation.

The existence of a hereditary predisposition has been indicated by clinical observation.

The possible environmental influence of prolonged constitutional disturbances and of pregnancy is suggested by clinical observation. Chronic inflammatory changes in the ear may sometimes act as an environmental factor necessary for production of the disease in the presence of a hereditary predisposition.

The histopathological appearance in some areas of an otosclerotic nodule indicates a resemblance to osteitis deformans, or Paget's disease, but differs greatly from the latter in its extent, rate of development and hereditary tendency. In neither is the etiology known, nor is any treatment known to have a favorable effect.

A definite relation is clinically evident between blue scleras, fragilitas ossium or osteogenesis imperfecta and otosclerosis. The existence of some common hereditary constitutional factor is suggested to account for predisposition to any one or any combination of these conditions.

The endosteal and endochondral layers of the capsule are resistant to most general bone diseases. Osteitis deformans, or Pagent's disease, and otosclerosis are exceptions. These two inner layers exhibit greater resistance to osteolytic processes. Infections and tumors invade and destroy the periosteal layer more readily, probably because of its air

7. Slaughter, E. G.: Experimental Hyperparathyroidism and Otosclerosis, Ann. Otol., Rhin. & Laryng. **49**:130-140 (March) 1940.

8. Lindsay, J. R.: Unpublished data.

cell content and marrow spaces. Infection which is blocked off in deeper parts of the temporal bone frequently does invade the endochondral layer, and sequestration of all layers sometimes occurs.

Death of the endochondral layer may result from electrocoagulation and is followed by active absorption along vascular channels with ultimate removal and replacement by new bone. While it has been possible experimentally to produce alterations in the bones of the skeleton and the outer layer of the labyrinthine capsule by various means the inner layers of the capsule have shown great resistance to change.

Further basic research into the biologic processes involved in normal cartilage and bone would seem indicated as a preliminary to the study of such bone diseases as osteitis deformans, fragilitas ossium and otosclerosis.

POSTNATAL REBUILDING AND OTOSCLEROTIC BONE FORMATION IN THE REGION OF THE OTIC CAPSULE

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THE REGION of the otic capsule, anterior to the oval window, which houses the fissula ante fenestram has long been regarded as a site of predilection for otosclerotic foci. Already in the development of this portion of the capsule there is evidence that it is a poorly formed and an unstable area, in that much intrachondrial and little perichondrial bone is produced. In addition, this area is traversed by the fissula ante fenestram, which is often of irregular shape and size. During late fetal and early postnatal life, various attempts to alter the size and shape of the fissula and rebuild the surrounding capsule have been observed. These often result in the blocking of parts or all of the fissula with cartilage masses or gnarly bone. A further attempt to correct such defects results in the resorption of the localized pathological cartilage or bone and the replacement of it by new bone. Sometimes such localized, circumscribed patches of new bone are porous and resemble the otosclerotic bone seen in the adult. The literature on these fetal and infantile changes need not be discussed here, since it is fully discussed in chapter 5 of Bast and Anson.[1]

PURPOSE

The purpose of this account is to correlate the abnormal contents of the fissula ante fenestram and the alterations in the surrounding otic capsule in the human fetus with abnormal and pathological processes in the fissular region of the adult human capsule, in order to determine what relation exists between such developmental pathological processes

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1. Bast, T. H., and Anson, B. J.: The Temporal Bone and the Ear. Springfield, Ill., Charles C Thomas, Publisher, 1949.

and otosclerotic bone. Observations on fetal as well as on adult otic capsules indicate that abnormal tissues in the fissular region are often seen, but the soft tissue within the main channel of the fissula ante fenestram is remarkably resistant to such pathological processes, especially otosclerotic bone. Yet, in those cases in which the fissula ante fenestram is unusually large or irregular the marginal cartilage often proliferates into those portions of the fissula which are extraneous to its main channel. Later ossification of such cartilages reduces the size of the fissula. In a smaller number of cases a cartilage mass may actually block the fissula, usually at or near its tympanic orifice. Ossification of such a cartilage mass may involve not only the cartilage but also the surrounding bone. The connective tissue of the fissula which had not chondrified is very resistant to such bony change. Thus, while the marginal cartilage of the fissular channel gives rise to cartilage masses which, in turn, may be changed into bone, the connective tissue of the fissula resists invasion by bone. That is to say, the fissular soft tissue does not initiate bony changes but attempts to preserve its integrity. The question that needs to be solved is whether these cartilaginous masses are eventually changed into a healed quiescent bone or whether they may initiate a progressive bony change known as otosclerosis.

MATERIAL

This account is based on serial sections of petrous bones obtained at autopsy in 38 cases. In most cases both ears were studied. The material was from the general run of autopsies and not from special cases of otologic disease. Although the results of otologic tests are available for some of the cases, there is nothing in the clinical records that is pertinent to the histological observations that I made on the temporal bones and the ears.

OBSERVATIONS

Abnormal areas were observed in the fissular region of the otic capsule in one or both ears of 25 of the 38 cases studied. In most of these the fissula ante fenestram appeared normal throughout its extent from the vestibule to the tympanum. In the majority of the cases (18) the abnormal tissue occurred along the cochlear margin of the fissula ante fenestram. Such changes, but with normal fissula, occur at all age levels, as shown in figures *2*, *3* and *4* from a *17* year old youth, *5* and *6* from a 40 year old man and *7* and *8* from men 62 and 72 years old, respectively. The frequency of this change along the cochlear margin of the fissula in the adult agrees pretty well with that in the fetus. In the fetus the pathological tissue was as a rule cartilage, although some bone occurred. In the present adult series these capsular changes were mostly bone, but some cartilage masses may still persist at any age (figs. *1*, *4*, *5* and *8*). These changes along with cochlear margin usually

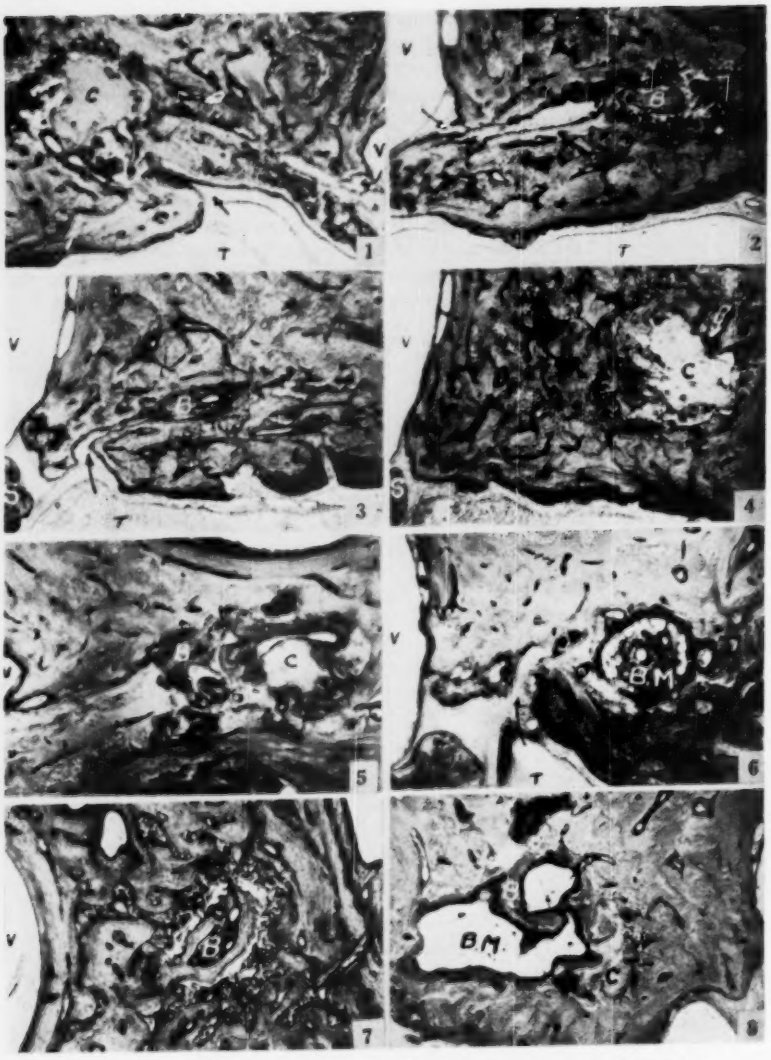

Plate I.—Figs. 1-8.—Sections through adult human petrous bones showing various pathological changes in the fissular region described in the text: *1*, from a youth aged 17; *2*, *3* and *4*, from a youth aged 17; *5* and *6*, from a man aged 40; *7*, from a man aged 62, and *8*, from a man aged 72. *C* indicates new cartilage; *B*, new bone; *BM*, bone marrow; *T*, tympanum; *V*, vestibule, and *S*, stapes. The opening of the fissula ante fenestram is shown at the arrow. × 28.

extend up into and involve the cupola of the fissula. In five cases only the cupola was involved, and the rest of the fissular region was unchanged. In six ears the fissula was blocked (figs. *9*, *10* and *11*),

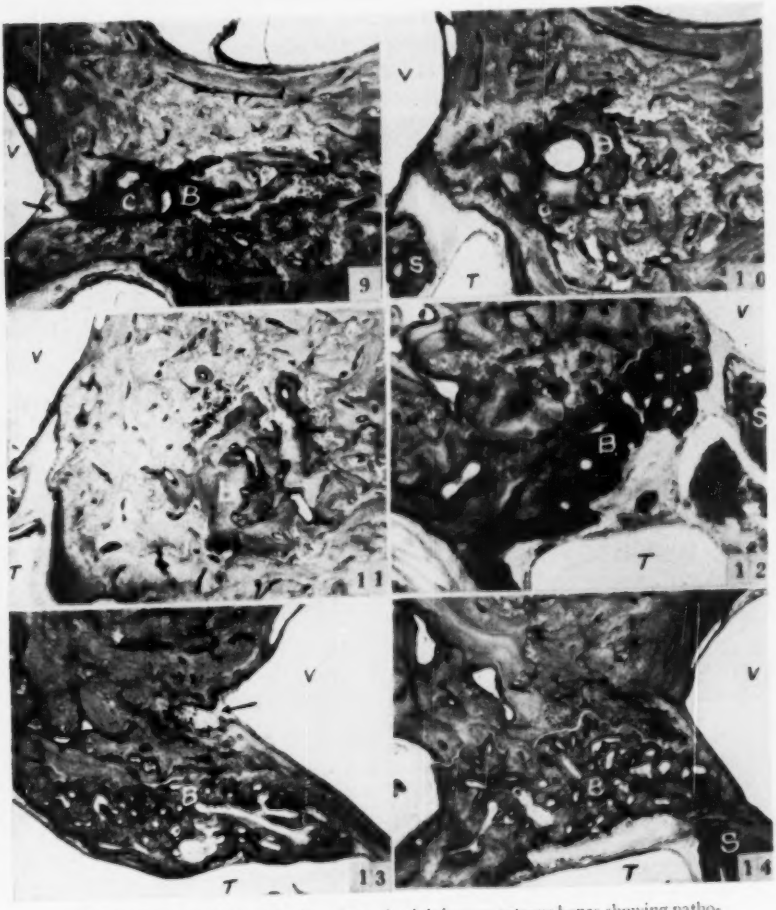

Plate II.—Figs. 9-14.—Sections through adult human petrous bones showing pathological changes in fissular area. Typical otosclerotic bone is shown in figures *12*, *13* and *14*; *9*, *10* and *11*, from a man aged 44; *12*, from a woman aged 31, and *13* and *14*, from a woman aged 21. *C* indicates new cartilage; *B*, new bone; *T*, tympanum; *V*, vestibule, and *S*, stapes. The opening of the fissula ante fenestram is shown at the arrow. × 28.

and in nine ears the new bone occurred at the tympanic margin (figs. *12*, *13*, *14*, and *15*).

Although our adult series is too small to permit conclusions, there is a definite indication that the pathological conditions in the fissular regions of adults correspond closely to those observed in fetuses and infants, both as to location and as to percentage.

HISTOPATHOLOGY

In the fetus most changes in the fissular region were cartilaginous, although some ossification was observed. In the adult most of the

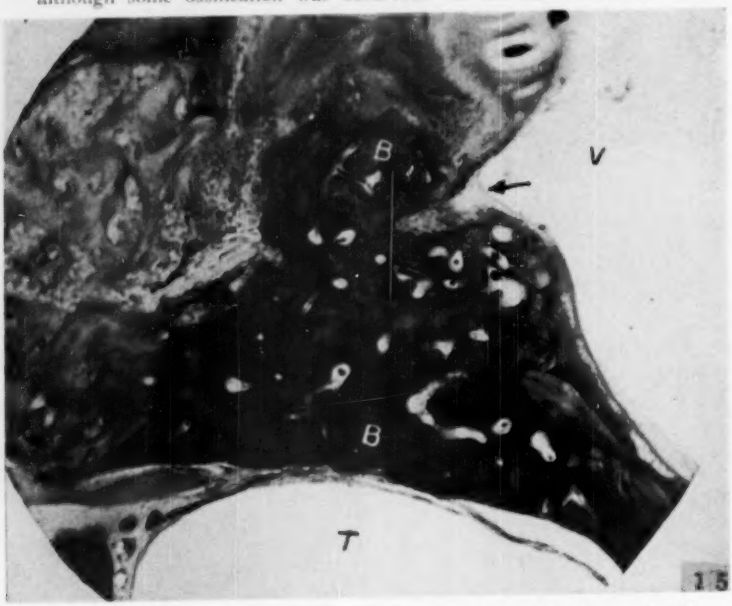

Plate III.—Fig. 15.—Section through human petrous bone showing otosclerotic bone (*B*) which has replaced most of the original bony capsule in the fissular region but has not destroyed the vestibular end of the fissula ante fenestram (at arrow). *B* indicates otosclerotic bone; *T*, tympanum, and *V*, vestibule. × 55. (N. W. Series, Dec. 6, 1929.)

changes were bony, although in a few cases the cartilage persisted. In the left ear of a 17 year old youth (fig. *1*) a cartilage mass like those seen in fetuses lies deep to the tympanic orifice of the fissula. In the right ear of the same youth such a cartilage mass was replaced by bone along the cochlear margin of the fissula and deep to the tympanic orifice (figs. *2* and *3*), but in the cupolar region (fig. *4*) the mass was cartilaginous and was in an active proliferating state. Such active

neoplastic cartilages have been noted in only a few fetal and infant ears. What becomes of these has not been determined. In a 40 year old man (figs. *5* and *6*), a 62 year old man (fig. *7*) and a 72 year old man (fig. *8*) such cartilage masses have been largely replaced by new bone or (figs. *6* and *8*) by a rim of bone and bone marrow.

A more extensive excavation and replacement with bone is shown in the case of a 44 year old man (figs. *9*, *10* and *11*). The vestibular end of the fissula is preserved, but much of the fissula is obliterated by cartilage and bone (fig. *9*). In the rest of the fissular region a definitely circumscribed mass of dense new bone with some blue mantles occupies the area (figs. *10* and *11*). Such circumscribed new bone as is shown in figures *2* to *11* appears to be healed, quiescent bone. Although it occurs in a circumscribed area, it does not show signs of progressive or invasive change and cannot be considered as active otosclerotic bone. In a small percentage of cases (four in the present series) definite patches of otosclerotic bone were observed (figs. *12*, *13*, *14* and *15*). It should be noted that no blue mantles are absent in all these cases. In all the cases observed this otosclerotic bone appears to arise in the capsule in the region of the tympanic orifice of the fissula ante fenestram and spreads in all directions. This bone is more porous and shows signs of continual rebuilding and extension. That the soft tissue filling the fissula is resistant to the invasion of the otosclerotic bone is shown in figure 15, where the new bone has surrounded the vestibular end of the fissula but has not replaced it.

COMMENT

It should be emphasized that this series of adult ears is too small to permit conclusions. It appears, however, that many of the bony changes observed are attempts to rebuild that part of the otic capsule which was left unstable by late fetal cartilage growths, especially in the fissular area. It is not clear why attempts to rebuild this seem to result in circumscribed masses of quiescent bone when the process occurs deep in the capsule, whereas when it occurs at the tympanic orifice of the fissula it seems to form active otosclerotic bone. A larger series of cases must be studied. The fact that some periosteal bone, which is less resistant to alteration, occurs along the tympanic surface may be a factor.

Again, the fact that blood vessels supplying this part of the capsule are normally found in the tympanic orifice of the fissula suggests that, owing to abnormal or unstable condition of this area, chemical substances may be liberated which stimulate these localized blood vessels to abnormal activity resulting in otosclerotic new growths.

CRITICAL EVALUATION OF ANTIHISTAMINIC DRUGS IN THE COMMON COLD

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ALTHOUGH it has been subjected to a burst of advertising pyrotechnics seldom seen in the field of medicine, the American medical profession is relearning once again the therapeutic maxim that there are no substances or combination of substances currently available which can be relied on to prevent or cure the common cold. It is not the first time that claims have proved to be groundless, but in this instance emotions have been aroused unnecessarily and tragedies have occurred which should have been avoided. Sufficient time has elapsed and enough valid data have been accumulated at leading medical centers in the United States and Great Britain to establish the fact that the common cold cannot be abolished by antihistaminic drugs. As the result of extravagant and irresponsible claims in behalf of antihistaminic drugs, the American public has been deluded into spending huge sums of money on the "sensational new discovery that kills colds in hours." It will continue to do so until it is educated by alert physicians to the foolishness of such practice.

The notion that allergy is mainly responsible for the common cold has never been substantiated by reliable investigators. Contentions that the common cold is an allergic response to contact with the cold virus [1] lie within the realm of hypothesis rather than within the domain of incontestable proof. Despite the knowledge that antihistaminic drugs are not viricidal, bactericidal or bacteriostatic, antihistamine therapy of the common cold has been based on an unproved assumption that an allergic component is involved in the production of the infectious common cold. The finding of histamine in the nasal secretions of persons with the common cold is often cited in support of the allergic mechanism of

From the Department of Otolaryngology, University of Illinois College of Medicine.

Read before the American Society of Ophthalmic and Otolaryngologic Allergy, Chicago, Oct. 7, 1950.

1. Fox, N., and Livingston, G.: Role of Allergy in the Epidemiology of the Common Cold, Arch. Otolaryng. **49**:575 (June) 1949.

colds. The technic employed by Troescher-Elam and associates [2] has been criticized by Feinberg and associates [3] as being uncontrolled; furthermore, the findings have not been substantiated.

That antihistaminic drugs are not innocuous is becoming progressively evident. In a survey of 247 physicians, 223 reported to Arbesman [4] the development of such side effects as drowsiness and dizziness among patients taking antihistaminic drugs for the common cold. Virtually all antihistaminic drugs possess sedative properties when given in moderate doses and are cerebral excitants when given in large doses. Other side effects that may occur are frequent urination, dryness of the mouth, insomnia, cardiac excitation, gastrointestinal disturbances, headache, fever, blurred vision, tinnitus, skin rash and nervousness. Instances have been reported of users of antihistaminic drugs falling asleep at work, while operating machinery or while driving automobiles. Children have died after the accidental ingestion of large doses of antihistaminic drugs. Such events are always tragic, but they are the more so when the drug is of no use for the condition for which it was purchased. These agents may have profound effects not only on the central nervous system and the genitourinary system but on the hemopoietic system as well, as made evident by hemolytic anemia and agranulocytosis. Crumbley [5] calls attention to the occurrence of hemolytic anemia in three patients who had received antihistaminic drugs over long periods; Drake [6] describes a case of agranulocytosis following the administration of methaphenilene (diatrin®) hydrochloride; Cahan and associates [7] and Hilker [8] report agranulocytosis due to tripelennamine (pyribenzamine®) hydrochloride. As a result of the over the counter sale of antihistaminic drugs, careless and habitual use may be expected, and the medical profession should anticipate serious reactions.

Treatment of the common cold is influenced by so many factors that only a rigidly controlled study eliminating as many variables as possible can be relied on for rational conclusions. Cautious investigators always

2. Troescher-Elam, E.; Ancona, G. R., and Kerr, W. J.: Histamine-Like Substance Present in Nasal Secretions of Common Cold and Allergic Rhinitis, Am. J. Physiol. **144**:711, 1945.

3. Feinberg, S. M.; Malkiel, S., and Feinberg, A. R.: The Antihistamines: Their Clinical Application, Chicago, The Year Book Publishers, Inc., 1950, p. 152.

4. Arbesman, C. E.: Correspondence, J. A. M. A. **143**:198 (May 13) 1950.

5. Crumbley, J. J., Jr.: Anemia Following Use of Antihistaminic Drugs, J. A. M. A. **143**:726 (June 24) 1950.

6. Drake, T. G.: Agranulocytosis During Therapy with the Antihistaminic Agent Methaphenilene (Diatrin®), J. A. M. A. **142**:477 (Feb. 18) 1950.

7. Cahan, A. M.; Meilman, E., and Jacobson, B. M.: Agranulocytosis Following Pyribenzamine, New England J. Med. **241**:865, 1949.

8. Hilker, A. W.: Agranulocytosis from Tripelennamine (Pyribenzamine®) Hydrochloride, J. A. M. A. **143**:741 (June 24) 1950.

concern themselves with such factors as the unreliability of subjective impressions; the suggestibility of both patients and physicians; the fluctuations of a patient's degree of immunity, and the difficulty of objective diagnosis of the common cold. Moreover, it is a common observation that a preponderance of nasal episodes regarded by the patient at first to be the common cold actually disappear within a few hours. A great many of the episodes involving sneezing and early nasal discomfiture never materialize into true infectious colds; for basically they involve transitory vasomotor changes in the nasal mucous membranes as the result of chilling, drafts, fatigue, emotional disturbances and menses. Spontaneous variation in the duration of the common cold was demonstrated years ago by Diehl.[9] It is because so many patients believe in the efficacy of any type of therapy that it is possible to convince the general public that almost any new treatment of the common cold is of value. Some of the reports from people receiving a placebo containing lactose could easily serve as admirable testimonials for this inert material.

The bulk of the published articles[10] hailing the use of antihistaminic drugs for the common cold have failed to separate the infectious cold from frequent vasomotor disturbances, nonspecific factors or allergic factors. Most of the reports did not indicate proper control series[11] and placebo therapy. Indeed, quite often the diagnosis of the common cold was not established beyond reasonable doubt[12] and patients with allergy were not adequately excluded. On the other hand, the failure of the antihistaminic drugs has been demonstrated in a series of carefully controlled studies in the United States and Great Britain. In this country reliable investigations have been conducted by various investigators independently at Cornell University, the Western Reserve University School of Medicine, the University of Minnesota, the University of Illinois College of Medicine, the Chicago College of Optometry, the United States Naval Training Station at Great Lakes, Ill., and the United States Military Academy at West Point. In Great Britain

9. Diehl, H. S.: Medicinal Treatment of the Common Cold, J. A. M. A. **101**:2042 (Dec. 23) 1933.

10. Brewster, J. M.: Benadryl As Therapeutic Agent in the Treatment of the Common Cold, U. S. Nav. M. Bull. **47**:810, 1947; Antihistaminic Drugs in the Therapy of the Common Cold, ibid. **49**:1, 1949; Antihistaminic Drugs in the Therapy of the Common Cold, Indust. Med. **18**:217, 1949; Treatment of Common Colds with an Antihistaminic Drug, Illinois M. J. **96**:302, 1949.

11. Gordon, J. S.: Antihistaminic Drugs in the Treatment of Upper Respiratory Tract Infection, Laryngoscope **58**:1265, 1948. Murray, H. G.: Treatment of Head Colds with an Antihistaminic Drug, Indust. Med. **18**:215, 1949.

12. Arminio, J. J., and Sweet, C. C.: Prophylaxis and Treatment of the Common Cold with Neohetramine (Thonzylamine Hydrochloride), Indust. Med. **18**: 509, 1949.

controlled experiments have been conducted under the auspices of the Medical Research Council at the request of the Ministry of Health.

In the study undertaken by Shaw and Wightman [13] at Cornell University a total of 443 students were under observation, one half receiving antazoline (antistine®) and the other half receiving a placebo of identical appearance. Sore throat, running or congested nose and malaise were aided equally by the placebo and the antihistaminic drug. These investigators questioned the beneficial effect of the antihistaminic drug in the treatment of the common cold.

The study at Western Reserve University School of Medicine by Feller and his associates [14] was undertaken to test the value of a group of antihistaminic drugs in the treatment and prevention of the common cold and undifferentiated respiratory diseases. The drugs used were thonzylamine (anahist,® neohetramine® and resistab®) hydrochloride, chlorprophenpyridamine (chlor-trimeton®) maleate, chlorprophenpyridamine maleate plus acetylsalicylic acid–acetophenetidin-caffeine formula (coricidin®), tripelennamine hydrochloride, diphenhydramine (benadryl®) hydrochloride, prophenpyridamine (inhiston®), chlorcyclizine (perazil) maleate and pyrilamine (neo-hist) maleate. Comparison of the duration of all treated and control illnesses showed that the antihistaminic drugs, regardless of dosage, had no beneficial effect. Likewise, there was no benefit either for the common cold or for the other undifferentiated respiratory illnesses. The duration of nasal symptoms was not abbreviated by administration of the antihistaminic agents. From the data gathered it was concluded that the antihistaminic drugs were ineffective as therapeutic agents for the common cold and other undifferentiated respiratory diseases.

At the University of Minnesota a controlled study was instituted by Cowan and Diehl [15] among students especially subject to the common cold for the purpose of evaluating the antihistaminic drugs as therapeutic agents. Five preparations were tested: tripelennamine hydrochloride, phenindamine (thephorin®) tartrate, phenindamine plus ascorbic acid, ascorbic acid and a placebo. Three hundred and sixty-seven students were treated during a four month period. In this study there was no indication that ascorbic acid alone, phenindamine alone, phenindamine

13. Shaw, C. R., and Wightman, H. B.: Study on the Treatment of the Common Cold with an Antihistaminic Drug, New York State J. Med. **50**:1109, 1950.

14. Feller, A. E.; Badger, G. F.; Hodges, R. G.; Jordan, W. S., Jr.; Rammelkamp, C. H., Jr., and Dingle, J. H.: Failure of Antihistaminic Drugs to Prevent or Cure the Common Cold and Undifferentiated Respiratory Diseases, New England J. Med. **242**:737, 1950.

15. Cowan, D. W., and Diehl, H. S.: Antihistaminic Agents and Ascorbic Acid in the Early Treatment of the Common Cold, J. A. M. A. **143**:421 (June 3) 1950.

plus ascorbic acid or tripelennamine had any important effect on the duration or severity of the common cold.

The study at West Point conducted by Hoagland and his co-workers [16] was confined to 190 young men in military service in whom head colds developed. Five preparations were employed: tripelennamine hydrochloride, chlorothen (tagathen®) citrate, a placebo, tripelennamine hydrochloride nasal spray and a placebo nasal spray. There was no significant difference in the proportion of cures reported by patients receiving oral antihistaminic drugs and those receiving oral placebos. Essentially the same proportion of patients received no benefit from either type of treatment. The effect of tripelennamine administered in the form of nasal sprays was essentially the same as the effect of inert materials given orally and intranasally.

Seal [17] carried out an extensive investigation at the United States Naval Training Station at Great Lakes, Ill., with 1,964 of the station personnel as subjects. One phase of the investigation was instituted at a time that the incidence of colds and other infections of the upper respiratory tract was high and all subjects were under constant medical observation. It was concluded that antihistaminic drugs did not effectively decrease either the number of men requiring attention at the dispensary or the number of admissions or in any way alter the symptoms among the patients. In a second phase of the Great Lakes study, investigation was made of the prophylactic value of the antihistaminic drugs among subjects who had been examined to eliminate those already having colds. In a third phase of the study, various persons were treated with an antihistaminic drug, a preparation containing atropine and a placebo. From the various group studies it was concluded that no evidence could be found to demonstrate that antihistaminic drugs are effective for the common cold.

In Great Britain clinical trials of antihistaminic drugs in the prevention and treatment of the common cold provided additional evidence that these agents are ineffective. Studies carried out under the auspices of the Medical Research Council [18] at the request of the Ministry of Health demonstrated that the antihistaminic drugs promethazine hydrochloride and chlorcyclizine hydrochloride had no value in the prevention of experimentally induced colds. A large scale clinical trial of thonzyla-

16. Hoagland, R. J.; Deitz, E. N.; Myers, P. W., and Cosand, H. C.: Antihistaminic Drugs for Colds: Evaluation Based on a Controlled Study, J. A. M. A. **143**:157 (May 13) 1950.

17. Seal, J. R.: cited in Drug Trade News, July 10, 1950, p. 36.

18. Clinical Trials of Antihistaminic Drugs in the Prevention and Treatment of the Common Cold: Report by a Special Committee of the Medical Research Council, Brit. M. J., 1950, Aug. 19, p. 425.

mine in widely separated areas in Great Britain and Northern Ireland [19] showed that this antihistaminic drug had little if any value in the treatment of the common cold. In addition, a large scale trial, involving 1,744 patients, carried out in eight factories and one public school in communities scattered throughout Great Britain failed to show that antazoline had any effect on the course of the common cold.

PRESENT INVESTIGATION

Initial Questionnaire.—Early in 1950 a study to assess the value of antihistaminic drugs in the treatment of the common cold was undertaken. Volunteer undergraduate subjects in attendance at the University of Illinois College of Medicine and at the Chicago College of Optometry participated in the investigation during a four month period. Shortly before the antihistaminic drug survey was begun, undergraduate members of each of the four years in attendance at both schools were asked to reply to a questionnaire so formulated as to supply data regarding the common cold pattern of each student. The questionnaire included the following questions: "Approximately how many colds do you get each year?" "What is the average duration of your cold?" "Does your cold start within the nose or with a sore throat?" "Have you ever had hay fever or asthma or hives?" "Do you believe there is any association between the onset of your colds and fatigue, change in weather and contact with people who have colds?" "In the past have you had any success in treating your colds?"

A substantial majority—479 University of Illinois medical students and 485 Chicago College of Optometry students—of the undergraduate bodies of the two institutions replied to the common cold questionnaire. Of the University of Illinois group, 475 students replied to the question "Approximately how many colds do you get each year?"; 477 College of Optometry students answered this question (table 1).

Approximately 79 per cent of the medical students and 78 per cent of the optometry students reported one to four colds a year, the remaining 21 and 22 per cent of both student bodies reporting more than four colds annually and in several instances as many as 12 colds a year. In general, the common cold pattern for the two student groups ran in a similar vein, with peaks in the number of colds occurring at one, one to two, two, two to three, three, three to four and four colds.

In response to the question "What is the average duration of your cold?" a reply was made by 476 medical students and 478 optometry students (table 2).

The average duration of the common cold was one week or less among 62.4 per cent (297) of the medical students and one to two weeks among 29.2 per cent (139) of the student body; it was believed to linger as a "cold" for from two to four weeks by 8.4 per cent (40) of the medical group. These values paralleled those of the optometry students;

19. Lorriman, G., and Martin, W. J.: Trial of Antistin in the Common Cold, Brit. M. J., 1950, Aug. 19, p. 430.

the average duration of the common cold was one week or less among 59.3 per cent (283) of this group and one to two weeks among 29.9 per cent (143); it was thought to linger from two to four weeks among 10.8 per cent (52) of the optometry group.

A similar parallelism was observed between the medical and the optometry group when they replied to the question "Does your cold start within the nose or with a sore throat?" Of a total of 470 medical students replying from the University of Illinois group, 59.7 per cent

TABLE 1.—*Data on Number of Colds Annually*

Number of Colds Annually	Number of Students	
	University of Illinois	Chicago College of Optometry
1	54	46
1-2	41	36
1-3	5	1
2	80	115
2-3	58	28
2-4	3	..
2-5	1	1
3	61	70
3-4	36	31
3-5	7	3
3-6	1	..
4	37	66
4-5	15	14
4-6	1	3
4-8	1	1
5	23	27
5-6	6	8
5-10	2	2
6	9	24
6-7	3	2
6-8	5	2
6-10	1	..
7-8	4	2
8	3	4
8-10	..	2
9	2	..
10	6	6
10-12	..	1
12	1	5

(281) asserted that colds began within the nasal cavity, 30.2 per cent (142) that they commenced in the throat and 10.1 per cent (47) that they started either in the nose, the throat or both. Of a total of 469 students replying from the Chicago College of Optometry group, 62.4 per cent (292) declared that colds began within the nasal cavity, 25.5 per cent (120) that they commenced in the throat and 12.1 per cent (57) that they started in either the nose or the throat or both.

The question "Have you ever had hay fever or asthma or hives?" elicited 470 answers from the medical students and 475 replies from the optometry undergraduates. Of the medical students, 23.2 per cent (109) gave a history of allergy, while only 15.8 per cent (75) of the optometry

students responded in like vein. The question "Do you believe there is any association between the onset of your colds and fatigue, change in weather and contact with people who have colds?" revealed that 70.1 per cent of the medical students believed fatigue to be a factor, 63.3 per cent declared that change in weather was a factor and 65.2 per cent asserted that there was an association between the onset of colds and

TABLE 2.—*Data on Duration of Colds*

University of Illinois		Chicago College of Optometry	
Number of Students	Average Duration, Days	Number of Students	Average Duration, Days
5	1-2	1	1
1	1-3	..	..
2	2	6	2
18	2-3	16	2-3
1	2-4	3	2-4
5	2-5	..	...
15	3	20	3
32	3-4	32	3-4
9	3-5	..	...
7	3-7	2	3-7
1	3-10	..	..
21	4	16	4
19	4-5	17	4-5
1	4-6	1	4-6
11	4-7	4	4-8
3	4-10	..	...
18	5	14	5
10	5-6	3	5-6
10	5-7	..	...
4	5-8	1	5-10
4	6	7	6
3	6-7	..	...
102	7	134	7
2	7-8	..	...
26	7-10	12	7-10
2	8	3	8-10
27	10	25	10
26	4-14	32	7-14
7	10-14	..	...
69	14	71	14
6	7-21	..	...
12	14-21	17	14-21
3	14-28	1	14-28
9	21	21	21
2	21-28	..	...
8	28	13	28

contact with people who had colds. On the other hand, fatigue was declared a factor by 74.1 per cent of the optometry students and change in weather by 81.1 per cent of the group, while 76.2 per cent stated that there was an association between the onset of their colds and contact with people who had colds. In this connection, it will be observed that substantially more optometry students than medical students believed that there was an association between the onset of colds and change in weather and contact with people having colds.

Replies to the question "In the past have you had any success in treating your colds?" showed a greater streak of conservatism among medical students than among optometry students. Of the medical students, 31.1 per cent reported previous success in treating their colds, whereas 45.8 per cent of the optometry students declared that they had previously been successful in the treatment of the common cold.

Clinical Investigation.—At the start of the clinical investigation of the antihistaminic drugs at the University of Illinois and the Chicago College of Optometry the medical school participants among the undergraduate body were requested to report to the department of otolaryngology outpatient clinic at the first sign of a cold. Here, reliance was placed not only on subjective diagnosis but also on objective nasal and pharyngeal findings made and recorded by examining physicians. At the optometry school, however, reliance was placed completely on the student's subjective diagnosis, and participants were accepted solely on the basis of their own interpretation of what constituted a cold. In short, an attempt was made to establish conditions descriptive of "medical diagnosis" and of uncontrolled "self diagnosis."

Each student participant was given an envelope containing a four day supply of antihistaminic drug or a placebo, with printed instructions to take the agent three times daily and with appropriate directions for discontinuing medication immediately when side effects, such as drowsiness or dizziness, occurred. Every effort was made to secure the best health interests of the students at all times. Neither the students nor the examining physicians were aware of the nature of the medicament or placebo. At the conclusion of the survey the various agents utilized in the clinical trial were revealed to me by a system of decoding. When a student's cold was terminated, he was asked to reply to a questionnaire regarding the efficacy or lack of efficacy of the therapy, the presence or absence of side effects and the clinical course of the cold.

The drugs and the placebo were given three times a day for four days. The daily doses were the following: thonzylamine, 25 mg.; tripelennamine, 50 mg.; methaphenilene, 50 mg.; chlorprophenpyridamine, 4 mg.; diphenhydramine, 25 mg., and doxylamine (decapryn®), 25 mg.

The medical group received its drugs disguised in uniform capsules, the optometry group receiving its medicinal agents undisguised and not contained in capsules. To insure that no bias would enter into the assessment of the results and to avoid making either the patient or the investigator aware whether antihistaminic drugs or placebos had been given in a particular case, the selection of the order of drugs and placebos for clinical utilization was first determined by means of random selection, with the one restriction that for each 24 students receiving antihistaminic drugs 12 students were to get placebos.

RESULTS

The investigation involved a total of 213 subjects, 139 students from the Chicago College of Optometry and 74 students from the University of Illinois College of Medicine. The therapeutic trials were carried out from early January to late April 1950. Of the 74 medical students, 10 received thonzylamine, 7 chlorprophenpyridamine, 7 tripelennamine, 9 diphenhydramine, 6 doxylamine, 8 methaphenilene and 27 the placebo. Of the 139 optometry students, 16 received thonzylamine, 17 chlorpro-

phenpyridamine, 16 tripelennamine, 13 diphenhydramine, 14 doxylamine, 18 methaphenilene and 45 the placebo.

Although all students were instructed to begin medication at the earliest sign of a cold, a number of them waited for several hours before

TABLE 3.—*Side Effects Attributed to Antihistaminic Drugs*

Drug	University of Illinois			Chicago College of Optometry		
	Students Receiving, No.	Students Reporting Side Effects		Students Receiving, No.	Students Reporting Side Effects	
		No.	Per Cent		No.	Per Cent
Neobetramine *	10	5	50	16	13	81.4
Chlor-trimeton *	7	3	42.8	17	11	64.7
Pyribenzamine *	7	4	57.1	16	15	93.7
Benadryl *	9	5	55.5	13	9	69.2
Decapryn *	6	4	66.6	14	11	78.5
Diatrin *	8	3	37.5	18	13	72.2
Placebo	27	9	33.3	45	22	48.8

* Registered trademark.

TABLE 4.—*Types of Side Effects Attributed to Treatment*

Main Symptoms Reported	Students Receiving Antihistamines		Students Receiving Placebo	
	Medical	Optometry	Medical	Optometry
Drowsiness	20	39	5	8
Headache	2	16	2	3
Fatigue	8	25	2	11
Diarrhea	3	..	1	..
Dryness of mouth	3	14	1	4
Itching	1	4	..	..
Constipation	3	6	..	..
Insomnia	1	..	..	..
Nervousness	1	8	..	3
Frequent urination	2	10	..	1
Trembling of hands	1	4	..	..
Pounding of heart	1	2	..	1
Dizziness	1	7	..	2
Blurred vision	..	4	..	..
Nausea or vomiting	..	1	..	..
Ringing in ears	..	2	..	..
Chills	..	3	..	1
Fever	..	8	..	3
Dryness of nose	..	..	..	2
Total	200		50	

commencing medication. Analysis of the data demonstrated that there is no validity to the contention that the antihistaminic drugs are more effective if taken within 12 hours after the start of a cold. Furthermore, the data revealed that antihistaminic drugs are no more effective than the placebo in aborting the common cold. In frequent instances, statements made in behalf of the placebo as an aborter of the common cold established the placebo as a more effective agent than the antihistaminic

drugs. For determining the effect of medication on the alteration of the common cold pattern of the students, as revealed in the initial questionnaire, the results of the therapeutic trials were divided into three classes: those which showed that the duration of the cold was less than half that of the previous colds, those which showed that it was only slightly shorter and those which showed that it was the same or longer than the duration of previous colds. Here, the superiority of the placebo was well established.

At this time it is interesting to call attention to the high incidence of side effects reported following treatment. In this connection, it appears that a higher incidence of side reactions may be produced by antihistaminic drugs during an acute infectious process such as the common cold than during an allergic nasal episode, as is encountered during an annoying siege of allergic rhinitis. Whether the phenomenon observed in the present investigation is constant is called to the attention of other investigators for study. The optometry students recorded far more side effects than did the medical students (tables 3 and 4).

COMMENT AND SUMMARY

Sufficient time has elapsed and enough convincing evidence has been accumulated at leading medical centers in the United States and Great Britain to establish the fact that despite the extravagant and irresponsible claims made, the common cold cannot be abolished by antihistaminic drugs. It is not the first time that claims for new cures of the common cold have proved to be groundless, but in this instance emotions have been aroused unnecessarily and the American public has been deluded into spending huge sums of money on a will-o'-the-wisp therapy. It will continue to do so until it is educated by alert physicians to the foolishness of such practice.

The notion that allergy is mainly responsible for the common cold has never been substantiated by reliable investigators. Contentions that the common cold is an allergic response to contact with the cold virus lie within the realm of hypothesis rather than within the domain of incontestable proof. Although antihistaminic drugs are neither viricidal nor bactericidal and are not bacteriostatic, antihistamine therapy of the common cold has been unwisely propagandized as being highly effective in the prevention and treatment of the common cold.

That antihistaminic drugs are not innocuous is becoming progressively evident. Virtually all antihistaminic drugs in moderate doses possess sedative properties and in large doses are cerebral excitants. Instances have been reported of users of these agents falling asleep while at work, while operating machinery or while driving automobiles. Children have died after the accidental ingestion of large doses of antihistaminic drugs. Such events are always tragic but are the more so when the drug is of no

use for the condition for which it was purchased. As a result of the over the counter sale of antihistaminic drugs, careless and habitual use may be expected and the medical profession should anticipate serious reactions.

Treatment of the common cold is influenced by so many factors that only a rigidly controlled study eliminating as many variables as possible can be relied on for valid conclusions. The bulk of the published articles hailing the use of antihistaminic drugs for the common cold have failed to separate the infectious cold from frequent vasomotor disturbances and from nonspecific factors or allergic factors. Most of the reports did not indicate proper control series and placebo therapy. The diagnosis of the common cold was not established beyond reasonable doubt, and patients with allergy were not excluded.

On the other hand, the failure of the antihistaminic drugs in the treatment of the common cold has been demonstrated in a series of carefully controlled studies in the United States and Great Britain. In Great Britain controlled studies conducted under the auspices of the Medical Research Council at the request of the Ministry of Health provided convincing evidence of the worthlessness of antihistaminic drugs in the prevention and treatment of the common cold. In the United States, reliable controlled studies have been conducted by various investigators working independently at Cornell University, the Western Reserve University School of Medicine, the University of Minnesota, the University of Illinois College of Medicine, the Chicago College of Optometry, the United States Naval Training Station at Great Lakes, Ill., and the United States Military Academy at West Point.

These carefully controlled studies conducted at the University of Illinois College of Medicine and at the Chicago College of Optometry demonstrate that antihistaminic drugs do not prevent, abort, shorten, curtail, reduce or stop the common cold. It was observed that antihistaminic drugs are no more effective than placebos in aborting a cold and that there is no validity to the contention that antihistaminic drugs are more effective if taken within a short time after the start of a cold. Attention is called to the high incidence of side effects attributed to treatment.

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CLASSIFICATION OF CHRONIC DISEASES OF NOSE AND ACCESSORY SINUSES

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THERE is no satisfactory classification of chronic nasal and sinus diseases. In most instances these conditions exist together, the nose being equally involved with the sinuses; hence a separate classification for nasal and for sinus diseases, respectively, is redundant. Attempts to label these diseases on the criteria of tissue changes, whether gross or microscopic, are difficult, because degenerative changes, such as hyperplasia, atrophy, cyst formation, suppuration and fibrosis, often exist together. Classification based solely on symptoms is similarly difficult because here, too, a symptom is often common to more than one condition.

The purpose of a classification is to identify a disease categorically so that it will be easily recognized as such by other practitioners. By combining a pathological condition which is more or less constant with a clinical term describing the dominant symptom presented by the patient, the following classification has been formulated.

- Atrophic rhinitis (sinusitis)
  - Primary
  - Secondary
- Suppurative rhinosinusitis (primary)
  - Bacterial
  - Fungous
  - Viral
  - Rickettsial
- Granulomatous rhinosinusitis
- Hyperplastic rhinosinusitis allergica
  - Seasonal
    - Manifest, or clinical
    - Borderline, or subclinical
      - Suppurative
      - Hyperesthetic
      - Vasomotor or obstructive
      - Neuralgic

Perennial
- Manifest or clinical
  - Suppurative
  - Hyperesthetic
  - Vasomotor or obstructive
  - Neuralgic
- Borderline or subclinical
  - Suppurative
  - Hyperesthetic
  - Vasomotor or obstructive
  - Neuralgic

Hyperplastic rhinosinusitis nonallergica
- Psychosomatic
- Endocrinal
- Physical
- Chemical
- Metabolic or nutritional

No attempt will be made to describe each disease in detail. Only enough information will be supplied to permit correct identification of the condition under discussion.

ATROPHIC RHINITIS

Some conditions are recognized as fixed entities, and so little trouble is encountered in properly placing them in a classification. This is true, for example, of atrophic rhinitis, which is generally recognized as being either of a primary or of a secondary type.

A. Primary.—Etiology: This disease entity has been known in the past under various names, such as "dry catarrh," "arteriosclerotic rhinitis," "sclerosing rhinitis." It is found so frequently in other members of the patient's family that it has come to be recognized as an inherited disease.[1] Evidence of its presence may be noted at birth. Ordinarily, primary atrophic rhinitis involves only the nose. Secondary infection may occur in the sinuses, but this is not the rule; hence the sinus mucosa is usually within the normal. Certainly the extreme crusting, exudation, atrophy, etc., are not seen in any of the sinuses except the exposed part of the ethmoid capsule.

1. Reichardt, H.: Zur Frage der Heredität der Ozaena, Monatschr. f. Ohrenh. **70**:389, 1936. Lermoyez, cited by Fleischmann, O.: Betrachtungen über die Ozaenagenese, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **133**:199, 1932. Schmidt, V.: Ozaena bei 7 jugendlichen Geschwistern, Monatschr. f. Ohrenh. **59**:1049, 1925. Baumgarten, E.: Über die Frühformen der Ozena, Arch. f. Laryng. u. Rhin. **22**:492, 1909. Fox, N.; Dinolt, R., and Morwitz, S.: Atrophic Rhinitis with Ozena: Etiologic Factors, Arch. Otolaryng. **33**:1033 (June) 1941.

Pathology: Changes occur in all parts of the mucous membranes and the underlying bone of the nose. There is a decrease in thickness of the epithelium accompanied with loss of cilia and a metaplasia from the normal respiratory type to a stratified squamous cell epithelium. The basement membrane is thinned out and frequently absent. The seromucinous glands degenerate and atrophy, with a resultant increase in viscosity of the secretions and a tendency toward crust formation. The tunica propria shows a marked fibrosis from infiltration of fibroblasts, plasma cells and lymphocytes, which are concentrated around the arterioles and veins. The lumen of the vessels becomes narrowed by the accompanying endarteritis and fibrosis. The periosteum becomes thickened, and an osteitic hyperplasia of the underlying bone occurs.

Symptoms: The chief symptoms are fetid odor, crust formation and nasal discharge. There is a marked widening of the nasal chambers which makes breathing quite free. A great amount of thick tenacious greenish discharge gives rise to hard dry crusts, which sometimes completely occlude the nose and may even form entire casts of the nasal cavity. There is an accompanying loss of the sense of smell. The nasal bridge is widened, causing a flattening of the face across the nasal bridge. The pharyngeal and laryngeal mucosa undergoes atrophy, causing associated symptoms. Roentgen studies of the sinuses fail to reveal any abnormalities.

B. Secondary Atrophic Rhinitis.—Etiology: This condition is characterized by a previous long-standing chronic suppurative process in the nose and sinuses. Though other members of the family are free from this condition, they frequently suffer from hyperplastic rhinosinusitis, suggesting that the latter condition is a precursor of atrophic rhinitis of the secondary type. Unlike the primary type, this disease is rarely, if at all, seen in children.

Pathology: The pathological changes are similar to those noted in the primary type of atrophy, except that the mucosa of the sinuses suffers equally with that of the nose. These changes consist in varying degrees of hyperplasia, atrophy, cyst formation, fibrosis and papillary hyperplasia. In general, these changes are less severe than those noted in the primary type of atrophy.

Symptoms: The amount of crusting and fetor is a great deal less in the secondary type of atrophic rhinitis. Although the nasal chambers are widened through atrophy of the soft tissues, there is no change in the architecture of the external bony skeleton. The typical facies of the primary type is absent, since this is primarily an adult disease with the severest changes occurring after the growth period. Roentgen studies reveal changes in the mucosa and underlying bone of the nasal sinuses.

SUPPURATIVE RHINOSINUSITIS (PRIMARY)

Etiology.—It is possible that as a result of trauma, or the presence of a foreign body, or after the acute exanthems of childhood or fungous, viral or rickettsial infections, a chronic infection may remain limited to the nose. If this does occur, it is rarely seen. In practically all cases of suppuration the condition exists equally in the nose and in one or more of the paranasal sinuses. In by far the majority of cases suppurative rhinosinusitis is secondary to an old allergic hyperplastic process which becomes secondarily infected. Primary suppuration of the sinuses does occasionally exist, however, and is seen most frequently following trauma to the antrum in a tooth extraction, or during the course of the acute exanthems or other infectious diseases.

Pathology.—Changes consist in hyperplasia, fibrosis, cystic formation and atrophy. These may occur in the same field, particularly when the condition is of long standing. The pathologist describes this tissue as a necrotic membrane. The epithelium may be normal in areas, but frequently a metaplasia is present so that the epithelium becomes flattened or entirely disappears. Numerous epithelial and subepithelial abscesses are present. The basement membrane is usually thickened. The substantia propria reveals the most intense changes. There is a tremendous infiltration of fibroblasts, round cells, polymorphonuclear leukocytes, histiocytes, plasma cells and other macrophages. The changes are similar to those noted in the hyperplastic tissues of the allergic sinus which has become secondarily infected, except that the edema of the stroma is less marked. The glands are frequently cystic and often filled with purulent material. Many are entirely fibrosed. The blood vessel walls become greatly thickened, and many of the thin-walled venous channels are entirely replaced by fibrous tissue. The underlying bone undergoes a condensing osteitis.

Symptoms.—Except for nasal discharge, chronic primary suppurative rhinosinusitis is usually asymptomatic. The diagnosis is made on the presence of free pus in the nasal gutters or positive roentgen demonstration of a fluid level in the sinus or the presence of certain organisms in the nasal or sinus contents on irrigation of the nose or sinus cavity. Only rarely is headache, fever, pain or any other symptoms referable to the sinus noted, though these symptoms will be present during an acute exacerbation following a cold. As a rule, chronic primary purulent rhinosinusitis is discovered during a search for etiological factors in persons suffering from some systemic disturbance such as arthritis, iritis or other disease. On the other hand, in the secondary type of chronic suppurative rhinosinusitis in which a previously hyperplastic process has existed, the symptoms associated with the allergy will have

been present before suppuration occurred. In addition, there will be physical and roentgen findings of purulence. These symptoms will be described later under the title "Hyperplastic Rhinosinusitis Allergica with Suppuration."

GRANULOMATOUS RHINOSINUSITIS

This includes all conditions of the nose and/or sinuses in which neoplasms or granulation tissue exists. These set up chronic specific inflammatory processes with characteristic cellular reactions and are identified by specific laboratory tests. They may be infectious or noninfectious, malignant or nonmalignant. Included among the noninfectious and infectious diseases are syphilis, tuberculosis, lupus, Boeck's sarcoid, yaws, rhinoscleroma, leprosy, leishmaniasis, glanders, anthrax, actinomycosis, blastomycosis, moniliasis, rhinosporidiosis, aspergillosis, leptothricosis, histoplasmosis, coccidioidomycosis, factitial lesions, eosinophilic granuloma and torulosis.

The tumors involving the nose and sinuses include fibromas, papillomas, adenomas, chondromas, angiomas, myxomas, neuromas, leiomyomas, rhabdomyomas, chordomas, myomas, teratomas, lipomas, plasmocytomas, xanthomas, carcinomas, sarcomas, and those of dental origin.

HYPERPLASTIC RHINOSINUSITIS

Hyperplastic rhinosinusitis presents the greatest difficulty of classification because of its numerous clinical, physical, and pathological vagaries. In the literature it has variously been known as intumescent rhinitis, hypertrophic rhinitis, polypoid rhinitis, cystic rhinitis, nonpurulent rhinitis and others.

General Pathology.—The disease is recognized grossly by various degrees of swelling and pallor, depending on the amount of edema present in the tissues. Between attacks the color of the tissues may be normal.

As these changes are part of a generalized change in the whole respiratory tract, they are, of course, bilateral. The greatest changes are particularly seen along the inspirating zone of the nose, namely, the middle meatus and its adnexa. Edema is more marked along the lower edge of the middle turbinate.

It is fair to say that when hyperplasia exists, an active irritant is responsible and is being contacted in amounts which are able to cause these changes. The changes usually correspond with results of the cytological examination. During an acute manifest attack, pallor and edema occur with eosinophils in the nasal smear, while during a low grade subclinical involvement hyperemia is prominent, and possibly a few

or normal numbers of eosinophils are seen in the nasal smear. Of the sinuses, the ethmoids become involved most frequently and most severely, though the changes are similar to those noted in the nose. Hyperplastic disease of the nose is prima facie evidence of the same condition throughout the membranes of the paranasal sinus group, and the pathological change is directly proportionate to the chronicity, the degree of sensitization of the person and the dosage of the shocking irritant.

In the subclinical or borderline type of hyperplasia, the nasal tissues do not reveal a great amount of swelling, and, accordingly, the changes are often overlooked. However, along the pendulous portions of the middle turbinates, edema is frequently noted. The anterior tip of the middle turbinate is often deformed and thickened. Enlargement of the posterior tips of the lower turbinates, the so-called mulberry type of turbinate, is often seen. There is usually a decided hyperplasia of the lymphoid tissue in the pharynx, especially in the person who has undergone tonsillectomy. When secondary suppuration has occurred, purulent material may be seen in the nasal gutters and plastered over the rostrum of the sphenoid.

Microscopically the epithelium undergoes cystic changes, most of the cells becoming secretory, i. e., goblet in character. Continued irritation results in a metaplasia to a flatter type of cell and infiltration with cellular elements, resulting in fibrosis. The basement membrane becomes exceedingly thick and infiltrated. The tunica propria shows the most striking changes. The cellular spaces become widened by edema, especially in the capillary zone next to the basement membrane. Continued edema in one area may result in herniation. This bulge, due to a lack of support of the stretched stroma, becomes a polyp. Secondary infection, as noted after a bout with an acute infection of the upper respiratory tract or when the person becomes highly allergic, causes polyps to develop more rapidly because of the added fluid in the tissues.

Cellular infiltration occurs. During an active allergic state a great number of eosinophils are seen, but during a period of inactive or low grade irritation the preponderant cell is the round cell and a good proportion of fibroblasts, histiocytes, leukocytes and plasma cells are also present. The blood vessels and lymph nodes undergo similar hyperplasia. The nodes become obstructed and cystic. Sometimes new ones are seen, but where the process is chronically active, the nodes tend to fibrose and to disappear entirely from the submucosa. Periarteritis develops in the vessels, and they may become entirely obstructed by fibrotic changes. There has been an attempt to differentiate the changes of the chronic allergic tissues from the chronic inflammatory tissues of

infectious origin. This is difficult because in most cases of chronic suppuration of the sinuses an allergic condition existed previously.

Bone changes also occur. It must be remembered that the mucosa acts as the periosteum of the underlying skeletal structure in the nose and sinuses. The submucosa is condensed in the area immediately over the bone. Here also lie the larger blood vessels. Obviously, changes in this tissue must influence the underlying bone. Fortunately, this bone has a haversian system which is able to augment the blood supply where epithelial injury has taken place. The continual osteoblastic and osteoclastic activities result in a condensation of the underlying bone.

Etiology and Symptoms.—Hyperplastic changes in the mucosa of the upper respiratory tract are characteristically found when this area becomes the shocking center in a person with an allergic diathesis. It is thought that similar changes occur in the mucosa of one who is not allergic but who is constantly exposed to other types of irritation, whether of a chemical, physical, endocrinous, metabolic or psychosomatic nature. Where hyperplasia exists, whether of mild or of extreme character, as in polyp formation, the allergic factor is more frequently responsible, though not always easily demonstrable.

Medical literature presents a confusing array of synonyms for the condition known as allergic rhinitis. When it occurs seasonally, the term "hay fever" is generally used, but when it is perennial numerous labels are affixed to this condition, each synonym being based on a description of the predominant symptoms. Characterized primarily by intermittent engorgement of the nasal mucosa, sneezing associated with free discharge of dilute mucus and nasal obstruction, this condition has paraded under the labels of vasomotor rhinitis, hyperesthetic rhinitis, pollinosis, hay fever and others. Many patients exhibit obstructive symptoms in the absence of sneezing. On the other hand, many patients sneeze constantly or intermittently without any great amount of nasal obstruction or discharge. Also, some patients exhibit these symptoms seasonally, and some exhibit them perennially. In addition, there are varying degrees of severity of symptoms, some being so mild as to go unrecognized. When less pathological change exists, less acute symptoms are present, and these are spoken of as being borderline or subclinical. The difficulties in properly evaluating the symptoms and pathological picture in subclinical cases have resulted in regimentation which falsely groups the subclinical cases into classifications which belie their allergic nature. Degrees of allergy must be recognized, just as are differences in severity of other diseases. With a lower degree of pathological change present, certainly fewer symptoms of allergy will be apparent. All these attributes, however, may be used descriptively for purposes of identification of the

particular phase of the disease under discussion. Thus, hyperplastic rhinosinusitis allergica may be seasonal or perennial. It may be of the frank or manifest type, or it may be of the borderline or subclinical type. The predominant symptom may be headache (neuralgic), sneezing (hyperesthetic), nasal obstruction or intumescence (vasomotor) or secondary suppuration (suppurative). Patients exhibiting any or all the above symptoms have in common the pathological entity recognized as hyperplasia, and hence all have actually the same disease. A description of each of the above phases of the common condition hyperplastic rhinosinusitis allergica and nonallergica follows.

HYPERPLASTIC RHINOSINUSITIS ALLERGICA

Seasonal.—Manifest or Clinical: The common name applied to the manifest seasonal type of hyperplastic rhinosinusitis allergica is "hay fever." Other terms include rose cold and pollinosis. The term "seasonal" denotes specifically the causational factor of a pollinosis which may be of tree, weed, flower or grass derivation. Because of the wide range of climate, the seasons vary, depending on the period of pollination of trees, weeds, flowers and grasses. The manifest type is easily recognized by the violence of its symptoms. It is characterized briefly by paroxysms of violent sneezing, feeling of fulness in the head, itching or burning in the nose, palate and eyes and a profuse stainless watery discharge. Tests with the proper antigens usually reveal the offending pollens. Elevated eosinophil counts are noted, in the blood and nasal secretions.

Borderline or Subclinical: The borderline type differs from the manifest type only in degree of severity; it lacks the violence noted in the latter condition. The nasal obstruction is less, as are the sneezing and nasal discharge. Because of the fewer symptoms, the condition is not generally recognized as being due to a pollinosis. The patients' dominant complaint may be headache (neuralgic), postnasal drip due to secondary infection (suppurative), intermittent nasal obstruction (vasomotor) or occasional sneezing bouts (hyperesthetic). It is usually associated with a more or less chronic cough and postnasal discomfort due to lymphoid pharyngeal irritation. The symptoms characteristically appear during the hay fever season and disappear when this season is over. They occur, of course, in persons who have a history of other allergies in themselves or in their families. Eosinophilia may not be present in the subclinical type of seasonal allergic rhinosinusitis.

Perennial.—Manifest or Clinical: The perennial manifest type of allergic hyperplastic rhinosinusitis presents symptoms similar to those noted in the seasonal type. There are frequent paroxysms of violent sneezing, with itching and burning of nose, palate and eyes. A profuse

stainless watery discharge is present during the paroxysms. Nasal obstruction is more or less constant and may be the predominant symptom (vasomotor or obstructive). Pain and tenderness over the sinal areas, as well as headache, may also be present (neuralgic). On the other hand, it may be the persistent sneezing (hyperesthetic) or postnasal discharge from secondary infection (suppurative) that causes the patient to seek relief. Pharyngitis with associated attacks of laryngitis, bronchitis and asthma is frequently noted. It continues, of course, throughout the year and is not influenced by seasons. It is commonly called "hyperesthetic or vasomotor rhinitis."

Borderline or Subclinical: The perennial manifest type of hyperplastic rhinosinusitis has a prototype in a borderline or subclinical syndrome differing from the latter only in degree of severity. It is frequently called "intumescent rhinitis." This is characterized by perennial symptoms of intermittent nasal obstruction or intumescence (vasomotor), occasional attacks of sneezing (hyperesthetic), lymphoid hyperplasia of the pharynx, frequently globi and chronic cough, frequent colds, nodes on the vocal cords, recurrent attacks of laryngitis, headaches (neuralgic), tubal tympanitis, mouth breathing, particularly at night, with resultant coated tongue, fetor oris, stomatitis and chronic purulent discharge postnasally (suppurative). A more detailed description of these symptoms has been given elsewhere.[2]

Careful observation of a large group of such patients over several decades has convinced us that those who initially present symptoms and findings of borderline or subclinical allergy become at some time frankly allergic. It is our feeling that the difference is merely one of degree, the milder stage being a quiescent state in a person with an allergic tendency, ready, when stimulated with the proper allergens, to develop into the extreme state of vasomotor imbalance manifested in frank allergic rhinitis.

HYPERPLASTIC RHINOSINUSITIS NONALLERGICA

Hyperplasia of the mucous membrane caused by extrinsic factors such as dust, pollens and bacteria occurs often. However, much evidence has been accumulated to prove that intrinsic factors may also cause continuous nasal irritation which ultimately results in hyperplasia. These are psychosomatic, endocrine, physical, chemical and metabolic disturbances. It is unnecessary to give a detailed description of this evidence, since the reader may obtain this information from the numerous

2. Fox, N.; Harned, J. W., and Peluse, S.: Borderline Allergy: Its Relation to Hyperplastic Disease of the Respiratory Tract, Arch. Otolaryng. **31**:502 (March) 1940.

articles which have appeared in the recent literature.[3] However, this type of hyperplastic rhinosinusitis does exist and should have a definite niche in a classification of chronic nasal and sinus diseases.

SUMMARY

A classification of chronic sinus and nasal disease provides a common terminology for discussion of these conditions. Since in most instances the nose and sinuses are simultaneously involved, a separate classification for each seems redundant. Though this classification combines both a pathological and a symptomatic terminology, it does fulfil the purpose of a classification, namely, to identify categorically each disease.

3. Walsh, T. E.: Vasomotor Rhinitis, Laryngoscope **60**:360, 1950. Fowler, E. P., Jr.: Unilateral Vasomotor Rhinitis Due to Interference with the Cervical Sympathetic System, Tr. Am. Laryng., Rhin. & Otol. Soc. **48**:139, 1942. Laub, G. R.: Rhinitis Vasomotoria Due to Imbalance of Endocrine Glands, Laryngoscope **55**:179, 1945. Saul, L. J.: Psychogenic Factors in the Etiology of the Common Cold and Related Symptoms, Internat. J. Psycho-Analysis **19**:451, 1938. Cone, A. J.: Sinus Disease in Children, Tr. Am. Acad. Ophth. **46**:79, 1941. Holmes, T. H.; Goodell, H.; Wolf, S., and Wolff, H. G.: The Nose: An Experimental Study of Reactions Within the Nose in Human Subjects During Varying Life Experiences, Springfield, Ill., Charles C Thomas, Publisher, 1950.

STREPTOMYCIN THERAPY IN ORAL TUBERCULOSIS

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SCHATZ, Bugie and Waksman [1] introduced streptomycin in January 1944 and reported the definite suppressive action of this new agent, among others, on cultures of Mycobacterium tuberculosis. In the same year Feldman and Hinshaw noted the therapeutic effect of streptomycin in experimental tuberculosis and followed this in 1945 with a preliminary report on the use of this antibiotic in the treatment of clinical tuberculosis.[2] Since that time the literature on streptomycin therapy has become voluminous. The indications for the use of streptomycin in pulmonary tuberculosis have become more or less uniformly standardized, and the favorable results of this treatment on tuberculosis of numerous extrapulmonary organs have been acknowledged.

However, while its beneficial action, particularly on lesions of the mucous membranes, has been observed, only very few reports of the therapeutic value of streptomycin or dihydrostreptomycin in oral tuberculosis have been published so far. This paucity of available data may justify the reporting of a small group of cases showing tuberculous involvement of the oral cavity. They came under our observation during a period of 2¼ years.

From the Veterans Administration Hospital.

Sponsored by the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the authors are the result of their own study and do not necessarily reflect the opinion or policy of the Veterans Administration.

1. Schatz, A.; Bugie, E., and Waksman, S. A.: Streptomycin: A Substance Exhibiting Antibiotic Activity Against Gram-Positive and Gram-Negative Bacteria, Proc. Soc. Exper. Biol. & Med. **55**:66 (Jan.) 1944.

2. Feldman, W. H., and Hinshaw, H. C.: Effects of Streptomycin on Experimental Tuberculosis in Guinea Pigs: A Preliminary Report, Proc. Staff Meet., Mayo Clin. **19**:593 (Dec. 27) 1944. Hinshaw, H. C., and Feldman, W. H.: Streptomycin in Treatment of Clinical Tuberculosis: A Preliminary Report, ibid. **20**:313 (Sept. 5) 1945.

Recognizing the considerable difficulty of separating cases when contiguous organs are involved, we wish to emphasize that this report concerns itself exclusively with tuberculosis of the lip, the oral mucosa, the tongue and the alveolar process. In other words, the more frequently found cases of tuberculous lesions involving the fauces and the tonsils, as well as lesions of the pharynx proper, have been purposely disregarded. They may become the subject of a later report.

Oral tuberculosis is a comparatively uncommon disease. The theories advanced for its rarity are various: the resistance of the thickened epithelium to the invading organisms; the protective and germicidal action of the saliva; the rich blood supply of this region; the constant movements of the tongue and cheek, preventing prolonged contact with the bacilli, and the antagonistic action of different bacteria among themselves.

The statistical data concerning the frequency of occurrence of oral tuberculosis vary somewhat with the different authors. Canuyt and others [3] state that tuberculosis of the mouth is rarer than tuberculosis of the pharynx in approximately the same relation as tuberculosis of the pharynx occurs less frequently than laryngeal tuberculosis. They also remark that tuberculosis is found pathologically more frequently than clinically.

Chiari [4] reported 12 cases (0.05 per cent) involving the pharynx, tongue and mouth and 12 others which showed tuberculous invasion of the tonsils among 24,410 patients observed during a period of five years. Cipes [5] in 1926 found four oral lesions among 2,800 tuberculous patients (0.14 per cent). Rubin [6] in 1927 reported 72 cases (1.44 per cent) involving the buccal membranes in 5,000 patients and occurring within 13 years. He found tuberculosis to involve the lips in six cases, the cheek, the gingiva and the floor of the mouth in nine, the pharynx in 22, the tonsils in 19 and the tongue in 16. Darlington and Salman [7] stated in 1937 that tuberculosis of the mouth is extremely rare. They described 10 lesions of the oral mucosa, 7 of which involved the gingiva in the form of ulcers.

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3. Canuyt, G.; Reverchon, Truffert, and Wild, C.: Les maladies du pharynx (clinique et thérapeutique), Paris, Masson & Cie, 1936, p. 48.

4. Chiari, O.: Über die Tuberculose der oberen Luftwege, Berl. klin. Wchnschr. **45**:984, 1899.

5. Cipes, B. J.: Oral Hygiene in Relation to Tuberculosis, Dental Cosmos **68**:50, 1926.

6. Rubin, E. H.: Tuberculosis of the Buccal Mucous Membrane, Am. Rev. Tuberc. **16**:39 (July) 1927.

7. Darlington, C. C., and Salman, I.: Oral Tuberculous Lesions, Am. Rev. Tuber. **35**:147 (Feb.) 1937.

According to Carmody,[8] the organs of the mouth are affected in the following order of frequency: (1) tongue tip, (2) tongue border and floor of mouth, (3) soft palate, (4) anterior pillar and uvula, (5) dorsum and base of tongue, (6) lower gums and jaw, (7) upper jaw and gums, (8) lips, (9) hard palate and (10) salivary glands. Reviewing the literature in 1915, Carmody [8] found that among 534 cases with 661 tuberculous lesions of the mouth, 277 lesions had occurred on the tongue, 97 on the palate, 51 in the pharynx, 48 on the tonsils, 48 on the upper jaw, 48 on the lips, 36 on the lower jaw and 16 on the cheeks. In 40 cases the location was not specified.

Vadala [9] reported in 1949 a single case of pharyngeal tuberculosis (0.025 per cent) occurring among 4,000 tuberculous patients at the Fitzsimons General Hospital during a period of two years. Oral tuberculosis was not mentioned.

All authors agree that clinical tuberculosis involves the tongue more frequently than any other organ of the oral cavity. According to Binstok,[10] lingual tuberculosis represents from 46 to 63.3 per cent of all cases of tuberculous lesions of the mouth. Morgagni in 1761 described a patient with "tubercles" of the tongue, but the first authentic case of lingual tuberculosis was reported by Portal in 1804 (cited by Morrow and Miller [11]). Virchow reported the first autopsy case in 1864 (cited by Farber, Friedland and Jacobs [12]). One of the oldest groups of statistics, published by Willigk,[13] mentions two cases of lingual tuberculosis among 1,317 patients (0.15 per cent). D'Aunoy and others [14] wrote in 1937 that a survey of the literature up to that time showed only 357 cases of reported lingual tuberculosis. The same authors noted that during the past 29 years (up to 1937) only five cases of lingual tuberculosis had been encountered among 730,524 admissions, of which 24,548 were for tuberculosis. Bryant [15] in 1939 found only nine cases (0.13 per cent) in 7,000 patients with far advanced pulmonary tuberculosis.

8. Carmody, T. E.: Oral Tuberculosis, Ann. Otol., Rhin. & Laryng. **24**:193 (June) 1915.

9. Vadala, A. J.: Streptomycin in Otolaryngologic Tuberculosis, Laryngoscope **59**:156 (Feb.) 1949.

10. Binstok, M. S.: La tuberculose de la langue: Sa clinique et sa thérapeutique, Rev. de. laryng. **58**:745 (July-Aug.) 1937.

11. Morrow, H. and Miller, H. E.: Tuberculosis of the Tongue, J. A. M. A. **83**:1483 (Nov. 8) 1924.

12. Farber, J. E., and Miller, D. K.: Beriberi Heart in a Tuberculosis Patient, Am. Rev. Tuberc. **51**:315 (April) 1945. Farber, J. E.; Friedland, E. L., and Jacobs, W. F.: Tuberculosis of Tongue: Report of 9 Cases, ibid. **42**:766 (Dec.) 1940.

13. Willigk: Prag. Vierteljahrsschrift, 1856.

14. D'Aunoy, R.; von Haam, E., and Connell, J.: Tuberculosis of the Tongue, Am. Rev. Tuberc. **35**:180 (Feb.) 1937.

15. Bryant, J. C.: Oral Tuberculosis, Am. Rev. Tuberc. **39**:738 (June) 1939.

Myerson [16] reported in 1944 that of 9,735 patients in a hospital for tuberculosis, 31 had this condition, a frequency of 0.32 per cent. Titche [17] in 1945 found 30 cases occurring among 8,204 admissions within 6½ years.

Katz,[18] as well as Schugt,[19] stated in 1941 that only about 400 cases of lingual tuberculosis had been reported up to that time and only 35 had been autopsied. Katz found 28 cases (19.9 per cent) of tuberculosis of the tongue in 141 consecutive autopsies at the Sea View Hospital. These findings are in contrast with the usual reports indicating that lingual tuberculosis occurs in only a small fraction of 1 per cent of cases clinically, but they confirm the observations of Canuyt and others [8] mentioned above.

Katz [18] found the majority of the lesions located at the base of the tongue. Valtan [20] saw the dorsum of the tongue most frequently involved. According to Brodsky,[21] the dorsum or the lateral border is the most usual seat of the disease. Carmody [9] reported the frequency of tuberculous lingual lesions in the following order: (1) tip, (2) border, (3) dorsum and (4) base. Titche [17] remarked that there is no definite site of predilection, though the lateral margins and the tip of the tongue appear to be most commonly involved by the disease.

Most authors agree that the largest number of tuberculous lesions of the tongue are found in white males. Only a very few cases of lingual tuberculosis in Negroes are known to have occurred. The incidence of the disease is about three times as great in the male as in the female (Padgett,[22] Rubin [6]); according to Martin,[23] the ratio is 6:1.

There is some disagreement about the age at which the greatest preponderance of tuberculous tongue lesions is observed. Katz,[18] as well as Morrow and Miller,[11] found the majority of their cases to occur

16. Myerson, M. C.: Tuberculosis of the Ear, Nose, and Throat: Including the Larynx, the Trachea, and the Bronchi, Springfield, Ill., Charles C Thomas, Publisher, 1944, p. 202.

17. Titche, L. L.: Tuberculosis of the Tongue, Am. Rev. Tuberc. **52**:342 (Oct.) 1945.

18. Katz, H. L.: Tuberculosis of Tongue, Quart. Bull., Sea View Hosp. **6**:240 (April) 1941.

19. Schugt, H. P.: Tuberculosis of the Tongue, Laryngoscope **51**:284 (March) 1941.

20. Valtan, O.: Intorno alle ulceri tubercolari della lingua e faringe, Riforma med. **45**:474 (April 6) 1929.

21. Brodsky, R. H.: Oral Tuberculous Lesions, Am. J. Orthodontics (Oral Surg. Sect.) **28**:132 (March) 1942.

22. Padgett, E. C.: Surgical Diseases of the Mouth and Jaws, Philadelphia, W. B. Saunders Company, 1932, p. 261.

23. Martin, W. F.: Primary Tuberculosis of the Tongue, South. Med. & Surg. **99**:348 (July) 1937.

in the second and third decades, whereas Baron and Boyd,[24] D'Aunoy and Miller,[25] Farber and others,[12] Finney and Finney[26] and Titche[17] encountered the greatest number in the fourth decade.

Some authors, for example, Martin,[23] reported primary tuberculosis of the tongue; but Myerson,[16] Padgett[22] and many other observers agree that tuberculosis of the oral cavity is always secondary to disease of the lung or to an extrapulmonary focus. Ornstein,[27] concurring, writes:

> Tuberculosis of the mouth and pharynx may be either a primary or secondary infection. Primary tuberculosis of the mouth and pharynx is so rare a condition, that we may regard all tuberculosis lesions found in the mouth and pharynx as secondary tuberculosis.

There are four theoretical ways by which the tubercle bacilli may reach the organs of the mouth from a primary lesion: by actual contact; by direct extension; by hematogenous seeding, and by lymphatic spread. It must be considered that, while in pulmonary tuberculosis bacilli are present in the sputum, this is not the case in extrapulmonary tuberculosis. Therefore, Ornstein[27] says:

> Though it would be simple to explain the infection as due to direct implantation of tubercle bacilli in abrasions in the mucous membranes of the mouth and pharynx during passage of tuberculous sputum, experience does not support the contention. Paradoxically, tuberculosis of the mouth and pharynx occurs infrequently in pulmonary tuberculosis with sputum heavily laden with bacilli. On the other hand, the complication of mouth and pharynx tuberculosis is frequently found in pulmonary tuberculosis in which there is difficulty in demonstrating bacilli in the sputum. Therefore is it not logical to assume that the involvement of the mouth and pharynx is through the hematogenous route?

Cipes[5] and Rubin[6] mention the poor hygienic conditions prevailing in the mouths of many patients suffering from tuberculosis; they comment on the infrequent appearance of oral tuberculous lesions in spite of these favorable circumstances.

Tuberculosis of the jaws is very rare (Chapotel,[28] Kremer and Wiese[29]). The commonest entrance of tubercle bacilli, in the presence of sputum positive for the bacilli, is through carious teeth, via the open pulp. Gross[30] explains the infection as being caused by addition of

24. Baron, B., and Boyd, L. J.: Tuberculosis of the Tongue, Bull. New York M. Coll., Flower & Fifth Ave. Hosps. **3**:152 (Oct.) 1940.

25. D'Aunoy, R., and Miller, J. W.: Tuberculosis of the Tongue: Report of 3 Cases, J. A. M. A. **95**:97 (July 12) 1930.

26. Finney, J. M. T., and Finney, J. M. T., Jr.: Tuberculosis of the Tongue, Surg., Gynec. & Obst. **40**:743 (June) 1925.

27. Ornstein, G. G.: Tuberculosis of the Mouth and Pharynx, Arch. Clin. Oral Path. **2**:26 (March) 1938.

28. Chapotel: Tuberculose mandibulaire, Rev. odont. **51**:444 (Nov.) 1930.

29. Kremer, W., and Wiese, O.: Die Tuberkulose der Knochen und Gelenke, Berlin, Julius Springer, ed. 8, 1930, p. 153.

30. Gross, H., in Berberich, J.: Lehrbuch der Mund- und Rachenkrankheiten, Leipzig, Georg Thieme, 1932, p. 327.

tubercle bacilli to a preexisting apical paradentitis. Brodsky [31] found 8 per cent of all periapical granulomas at the Sea View Hospital to be tuberculous in nature. Thoma [32] lists three types: (1) peripheral or ulcerating infection (the most frequent form in the alveolar process); (2) central or osteitic infection, and (3) diffuse or osteomyelitic form. This is the most serious manifestation. Tuberculosis of the upper and of the lower jaw run somewhat different clinical courses. While tuberculosis of the mandible is an aggressive disease, tuberculosis of the maxilla runs a relatively mild course. Tuberculosis of the upper alveolar process is said to assume a middle road in aggressiveness (Padgett [32]).

The types of tuberculosis of the oral mucosa (in addition to the rare lupus vulgaris) include, according to Brodsky,[31] the following forms: (1) ulcer, (2) fissure, (3) granuloma, (4) tuberculoma and (5) glossitis. The ulcer is by far the most frequent and, according to Herrmann,[33] also the severest type. It shows undermined edges, has an irregular outline and is often covered with a grayish exudate. It lacks the induration of cancerous lesions. The histological features of tuberculous lesions are too well known to be mentioned here. They will be presented later in some of the biopsy reports of our cases.

Among the symptoms of the disease, by far the most outstanding one in the presence of ulceration is pain, especially on deglutition.

The diagnosis depends on biopsy.

The prognosis of all tuberculous infections of the mouth heretofore has been poor, especially in the presence of ulcerations. Granulomas gave a somewhat better outlook. Once established, the lesions develop independently of the progress of the pulmonary disease. Ornstein [27] remarks, "Contrary to what has been previously written, the prognosis depends more upon the control of the lesions in the mouth than on the underlying pulmonary tuberculosis which does not frequently run an active course."

Before the advent of the antibiotics, the treatment of tuberculous lesions in the mouth had usually been palliative. Caustics or direct cautery have been applied locally, sometimes with surgical excision of the lesions preceding these. Ultraviolet rays, radiation from a cold quartz lamp, radium and roentgen radiation, especially in the form of grenz rays, have been used with some success, as was tuberculin therapy. In terminal cases local anesthetics have been employed and infiltration or sometimes resection of the lingual nerve has occasionally become necessary to control the pain.

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31. Brodsky, R. H.: Tuberculosis of the Mouth, Ann. Dent. **6**:167 (Sept.) 1947.

32. Thoma, K. H.: Oral Pathology: A Histological, Roentgenological and Clinical Study of the Diseases of the Teeth, Jaws and Mouth, ed. 2, St. Louis, C. V. Mosby Company, 1944, p. 1251.

33. Herrmann, F.: Tuberculose der Mundschleimhaut, in Berberich, J.: Lehrbuch der Mund- und Rachenkrankheiten, Leipzig, Georg Thieme, 1932, p. 455.

Our own series consists of 11 oral lesions of proved tuberculous origin which occurred among 1,796 admissions (0.61 per cent). Of these, five (0.28 per cent) occurred on the buccal mucosa, three (0.17 per cent) involved the tongue, two (0.11 per cent) were located on the alveolar process and one (0.06 per cent) was on the lower lip.

Superficial and deep ulcers, as well as granulomas, predominated in our cases. In addition, we observed two fistulas. One penetrated from the alveolus into the gingiva and the other from the buccal mucosa through the skin of the cheek. One nonlingual lesion assumed the form of a fissure.

All our lesions were secondary to far advanced pulmonary tuberculosis, and all occurred in white male adults. Every lesion was verified by biopsy to be of tuberculous origin, except that of the buccal fistula, in which tubercle bacilli were isolated from the drainage.

In most of our cases the diagnosis was made during routine otolaryngological or dental examinations. Only when there was ulceration present did the patients complain of pain. One patient ascribed the sensation of a "foreign body" in the mouth to irritation from a dental plate. In none of our cases were the regional lymph nodes swollen or painful.

In view of the rare incidence of oral tuberculosis, it is not surprising that there exist only a few reports regarding the use of streptomycin or dihydrostreptomycin for treatment of this condition.

Shamaskin [34] treated three patients with lingual tuberculosis in 1947. Under daily intramuscular therapy with 2.0 Gm. of streptomycin the ulcers in his first patient healed after 60 days and had remained healed for three months. Treatment was continued for three months after the ulcers had disappeared. His second patient was treated topically with 0.5 Gm. of streptomycin for 30 days, but the dosage was then changed to 2.0 Gm. of streptomycin given intramuscularly because local therapy was unsatisfactory. While the oldest ulcer on the dorsum of the tongue was very much improved after 100 days of this regimen, relapse occurred and it assumed almost the same size as at the beginning of treatment. A blister was later observed on the under surface of the tongue. A third patient received combined therapy with 0.5 Gm. of streptomycin topically and 2.0 Gm. intramuscularly daily. The ulcers were five years old. Administration of streptomycin was abandoned after 60 days because the lesions did not respond to treatment and the patient's condition continued to deteriorate. Shamaskin wondered about the role the age of tongue lesions might play in their susceptibility to the effects of streptomycin. His conjecture is that "in tuberculosis of the tongue there

34. Shamaskin, A.: Tuberculous Ulcers of the Tongue: Three Cases, Am. Rev. Tuberc. **56**:419 (Nov.) 1947.

might exist the same condition as in pulmonary tuberculosis, namely, the older the lesions the more there is fibrosis and the less success with streptomycin will be obtained."

In 1948, Wolfer and others [35] reported as the first instance of streptomycin therapy in lingual tuberculosis a case in which streptomycin was successful in curing an ulcerative lesion located on the central part of the dorsum of the tongue. The treatment consisted in administration of a total of 75 Gm. of streptomycin in daily doses of 1 Gm. intramuscularly supplemented by local infiltration for a short time. The ulcer healed but had not responded to local infiltration.

While there exist some reports (Hinshaw and others,[36] Pfuetze and others,[37] Sweany [38] and Waksman,[39] and others [39a]) mentioning the beneficial effect of streptomycin on oropharyngeal lesions and on mucous membranes in a general way, the only other data on streptomycin therapy of oral tuberculosis that we were able to find appeared in the minutes of the several Veterans Administration Streptomycin Conferences.[40] Unfor-

35. Wolfer, H.; Hirshleifer, I., and Shapiro, R. N.: Treatment of Ulcer of the Tongue with Streptomycin, J. A. M. A. **136**:249 (Jan. 24) 1948.

36. Hinshaw, H. D., and Feldman, W. H.: Streptomycin in Treatment of Clinical and Experimental Tuberculosis, Ann. New York Acad. Sc. **48**:175 (Sept. 27) 1946. Hinshaw, H. C.; Feldman, W. H., and Pfuetze, K. H.: Streptomycin in Treatment of Clinical Tuberculosis, Am. Rev. Tuberc. **54**:191 (Sept.) 1946; Present Status of Streptomycin in Treatment of Tuberculosis, Connecticut M. J. **11**:247 (April) 1941.

37. Pfuetze, K. H.; Glover, R. P.; White, E. F., Jr., and Feldman, W. H.: Clinical Use of Streptomycin in Treatment of Tuberculosis, Dis. of Chest **12**:515 (Nov.-Dec.) 1946. Pfuetze, K. H., and Pyle, M. M.: The Use and Misuse of Streptomycin in the Treatment of Tuberculosis, Journal-Lancet **68**:431 (Nov.) 1948.

38. Sweany, H. C.: The Value of Streptomycin in the Treatment of Tuberculosis, Dis. of Chest **15**:631 (June) 1949.

39. Waksman, S. A.: Antibiotics and Tuberculosis: A Microbiologic Approach, J. A. M. A. **135**:478 (Oct. 25) 1947; Streptomycin: Nature and Practical Applications, Baltimore, Williams & Wilkins Company, 1949, p. 308.

39a. McDermott, W.: Streptomycin in the Treatment of Tuberculosis, J. Nat. M. A. **41**:167 (July) 1949. Streptomycin in the Treatment of Tuberculosis, American Trudeau Society, Report of Clinical Subcommittee, Am. Rev. Tuberc. **59**:106 (Jan.) 1949.

40. (*a*) Minutes of the Third Streptomycin Conference, May 1-3, 1947, United States Veterans Administration, St. Louis, p. 111. (*b*) Minutes of the Fourth Streptomycin Conference, Oct. 9-12, 1947, United States Veterans Administration, St. Louis, Veterans Administration, Branch Office No. 9, p. 58. (*c*) Minutes of the Fifth Streptomycin Conference, May 15-19, 1948, United States Veterans Administration, Chicago, Veterans Administration Branch Office No. 7, Tuberculosis Section, p. 132. (*d*) Minutes of the Sixth Streptomycin Conference, Oct. 21-24, 1948, United States Veterans Administration, St. Paul, Minn., Veterans Administration, Branch Office No. 8, Tuberculosis Section, p. 121. (*e*) Minutes of the Seventh Streptomycin Conference, April 21-24, 1949, United States Veterans Administration, Washington, D. C., Veterans Administration, Area Office, p. 263.

tunately, in the later summaries these lesions are grouped together as oropharyngeal tuberculosis, making a differentiation into lingual, buccal, alveolar, tonsillar, faucial or pharyngeal tuberculosis impossible.

At the conference of May 1947 [41] eight cases of tuberculosis involving the tongue and tonsils were reported as treated with streptomycin. In five of the cases the glossitis or tonsillitis became healed; in two it improved, and in one, a terminal case, it was unaffected.

This summary was followed at the time of the Eighth Conference, in October 1947,[40b] by report of 11 lingual cases (of which six were healed, four were improved and one was unchanged under streptomycin therapy) and two cases of involvement of the oral mucosa, both of which were healed.

At the Fifth Conference,[42] in April 1948, data on 38 ulcerative lesions of the tongue, buccal mucosa, tonsils or pharynx were collected. Of these, 36 were cured, one was improved and one was unchanged or relapsed under treatment. In some of the cases of oropharyngeal tuberculous diagnosis was made by biopsy, but a characteristic lesion in the presence of sputum proved to be positive for acid-fast bacilli by culture was regarded as acceptable evidence of tuberculous causation. (It may be mentioned here that we observed, in addition, a case of granuloma of the middle left margin of the tongue and another of dental granuloma—both granulomas indistinguishable from a tuberculous process—in the presence of sputum positive for tubercle bacilli. However, as biopsies revealed the granulomas to be of apparently nonspecific origin, we did not include these two cases in our report.)

At the Sixth Conference,[40d] in October 1948, a total of 49 oropharyngeal lesions were reported. Of these, 40 (81.6 per cent) were cured, 8 (16.3 per cent) were improved and 1 (2.0 per cent) was unchanged under streptomycin therapy.

At the Seventh Conference,[40e] in April 1949—the latest data available to us—54 hospitals reported a total of 56 oropharyngeal lesions; of this number, 47 (82 per cent) were cured, 9 (14 per cent) were improved, 1 (2 per cent) was unchanged, 2 (4 per cent) had become worse and 5 (9 per cent) had relapsed.

41. Report of Committee on Therapy and Its Subcommittee on Streptomycin, American Trudeau Society, Am. Rev. Tuberc. **56**:477 (Nov.) 1947; Effects of Streptomycin on Tuberculosis in Man, Council on Pharmacy and Chemistry, J. A. M. A. **135**:634 (Nov. 8) 1947. Footnote.[40a] Streptomycin in the Treatment of Tuberculosis: Current Status: Report to the Council on Pharmacy and Chemistry, J. A. M. A. **138**:584 (Oct. 23) 1948. Footnote [40c]

42. Riggins, H. McL.; Lincoln, N. S.; Horton, R.; Stokes, A. M., and Monroe, J.: Streptomycin and Tuberculosis: Dihydrostreptomycin in Pulmonary Tuberculosis, Tr. Nat. Tuberc. A. **45**:99, 1945.

At these conferences the consensus was that excellent results were obtained with streptomycin therapy. It was stated that the method of topical application of this antibiotic was abandoned as unsatisfactory. Therefore, we have used intramuscular therapy exclusively.

If we assume that all cases reported of oropharyngeal tuberculosis do represent individually new instances of tuberculosis and if we further estimate with Binstok [10] and Carmody [8] that in about 50 per cent of the cases the lesions occur on the tongue, with a large percentage localized in the fauces, tonsils and pharynx, it will become apparent that the incidence of buccal and alveolar tuberculosis is small, indeed, and tuberculosis of the lip is extremely rare.

REPORT OF CASES

CASE 1.—L. C., a 51 year old white man, was admitted to the Veterans Administration Hospital with far advanced bilateral active pulmonary tuberculosis of the reinfection type. Bilateral cavitations and bilateral chronic fibrinous pleurisy were present. The sputum was positive for tubercle bacilli.

Previously tuberculosis of the right lingual margin had been diagnosed elsewhere by biopsy. It had been treated unsuccessfully with 25 per cent silver nitrate and Finsen light.

When he was first seen by one of us (H. O.) on Nov. 18, 1947, the following findings were recorded:

1. An ulceration existed at the junction of the anterior and middle third of the right lingual margin. No induration was present.

2. Edema of the uvula and ulceration of the base of the uvula extending into the left posterior pillar were noted.

3. An extensive tuberculous edema with infiltration of the larynx developed, consisting of a tremendous swelling of the left arytenoid and infiltration of the left true and false cords and the interarytenoid area. The right vocal cord was slightly reddened. The patient was unable to talk above a whisper.

4. Chronic purulent otitis media, tuberculous in origin, was present on the right side, causing impaired hearing, which later developed into complete deafness.

The first streptomycin regimen was started on November 29 and completed, after 120 days, on March 16, 1948. The streptomycin was given intramuscularly, 0.5 Gm. twice daily.

On Dec. 9, 1947 the ulcer of the tongue had healed with scar formation. The left pillar and the uvula were normal. The larynx was improving; the right cord was nearly normal, but edema of the left arytenoid was still present, though somewhat reduced. There was less reddening and swelling of the left true and false cords.

On December 16 the right ear perforation was healing.

On March 8, 1948 the vocal cords were normal. The swelling of the left arytenoid area was still present but considerably reduced in extent. The patient was talking with a normal voice.

The perforation of the right ear had healed completely.

At the end of the 120 days of streptomycin therapy the patient had become afebrile. He had gained 27 pounds (12.2 Kg.), but the sputum was still positive for the bacilli on direct smears. Roentgenograms did not show a change in the lungs.

On May 30, 1949 streptomycin was given again because of recurrence of the lingual ulcer. This time the patient received 1.0 Gm. of streptomycin intramuscularly. Its use was discontinued after 44 days because many red blood cells were found in the urine. Long before the end of the 44 days the ulcer of the tongue had completely healed and has remained so up to the present time, at which only a small scar can be discerned on the right lingual margin.

On June 2, 100 per cent of the bacilli were resistant to 1 microgram of streptomycin, 100 per cent were resistant to 10 micrograms, and 50 per cent were resistant to 100 micrograms. On September 8, 100 per cent were resistant to 1, 10 and 100 micrograms.

In February 1950 a diagnosis of tuberculosis of the left kidney was made, based on roehtgen findings and demonstration of tubercle bacilli in the urine.

Comment.—This patient showed evidence of severe and generalized dissemination of tuberculosis. Presumably the disease spread by the hematogenous way. The ulceration of the tongue had disappeared after only 10 days but recurred after 18 months. Since the second course of streptomycin, the ulcer has remained healed, and there has been no recurrence up to the present time. In retrospect, it may be said that the hematuria leading to the discontinuation of the second streptomycin course was probably the first sign of renal involvement, which was later substantiated by roentgenographic and bacteriologic means.

Case 2.—H. Y., a 38 year old white man, was admitted for far advanced bilateral active pulmonary tuberculosis of the reinfection type. His sputum was positive for tubercle bacilli.

On Nov. 12, 1947 an area in the middle third of the left dorsal margin of the tongue showed granulomatous tissue. The patient stated that five months earlier at another hospital a biopsy specimen had been taken from this area. At that time a cancer was suspected, but it could not be substantiated by biopsy. When first seen here, he refused another biopsy, but as some irritation developed in this area of the tongue he consented to a biopsy two months later. The pathological report of Dr. S. Rifkin, our pathologist (who also examined our other specimens) read, "One section of the block shows a moderate-sized area of conglomerated tubercles with at least six giant cells, the whole being surrounded by a well formed capsule and lying just below the mucosa. The impression is tuberculosis."

Streptomycin was given intramuscularly, 1.0 Gm. daily from Feb. 12 to May 21, 1948. The lingual lesion had healed after about four weeks. Streptomycin sensitivity at that time was 0.5 micrograms per cubic centimeter. He received a disciplinary discharge May 22.

On July 8, 1949 he reentered this hospital because of his pulmonary tuberculosis. At that time examination showed a scar on the left middle dorsal margin of the tongue, with no activity. His tongue has been normal since that time. His sputum remained positive for tubercle bacilli.

Comment.—Elsewhere the tuberculous lesion of the tongue was originally thought to be of malignant origin. However, the first biopsy did not show any pathologic condition.

The tuberculous lesion has remained healed up to the present time (more then two years). It is possible that the complete lingual focus was removed by our biopsy, as evidenced histologically by the well formed capsule around the lesion.

CASE 3.—H. F., a 54 year old white man, entered this hospital with far advanced pulmonary tuberculosis of the reinfection type and bilateral cavitations. His sputum was positive for tubercle bacilli.

He stated that in two other hospitals three biopsies of his tongue had been done previously. The first one had shown subacute inflammatory changes but no neoplasm. A second one had shown squamous cell carcinoma of the tongue, grade 2, for which he had received radium therapy. He was then transferred to another hospital, where a diagnosis of tuberculosis of the tongue was established by biopsy and later verified. For this condition he had received 0.5 Gm. of streptomycin intramuscularly four times daily for six weeks.

On the first examination here a scar in the middle of the posterior half of the tongue was noted and diagnosed as inactive tuberculosis. Later examination revealed a tuberculous crater with beginning reactivation in the middle of the posterior half of the tongue surface. The patient refused a biopsy here. The mucosa of the mouth, pharynx and larynx was normal.

On May 13, 1949 administration of streptomycin intramuscularly, 1.0 Gm. daily, was started, but its use was discontinued on June 1 because of the development of a spontaneous horizontal rotatory nystagmus of the first degree to the left side together with severe dizziness, nausea and vomiting. His hearing remained normal. The patient stated that a similar reaction had occurred during his first course of streptomycin therapy elsewhere.

His tongue lesion was healed on June 1 and has remained so up to the present time. All vestibular symptoms disappeared five days after the discontinuation of use of the drug. His hearing and reaction to the caloric test are normal at the present time. No change has occurred in his lungs. Twenty-five per cent of the bacteria were resistant to 100 micrograms, 25 per cent were resistant to 10 micrograms and 50 per cent were resistant to 1 microgram.

Comment.—This is the only patient in our present series in whom there developed—for the second time—a vestibular reaction after streptomycin therapy. His symptoms disappeared five days after streptomycin administration had been stopped. His hearing and vestibular reaction have remained normal. The tongue lesion has shown no recurrence for 11 months.

CASE 4.—R. L. C., a 53 year old white man, was admitted for far advanced active bilateral pulmonary tuberculosis of the reinfection type. His sputum was positive for tubercle bacilli at the beginning of our observation and remained so until three months after his oral lesion had healed. The sedimentation rate was 22 mm.

In November 1948 all remaining teeth were extracted. In April 1949 dentures were constructed. In May he complained of pains in the region of the lower left cuspid which he attributed to irritation from pressure of his new denture. Examination of this area revealed a lesion on the lower gum at about the side of the left cuspid, consisting of a fistulous opening with purulent secretion. Roentgenographically a V-shaped loss of bone was noted in the alveolar process of this area.

Biopsy of the gingiva from this location on May 25 revealed a tuberculous process.

Administration of 1.0 Gm. of streptomycin intramuscularly daily for 90 days was initiated on June 4. His treatment was twice interrupted for a short time, each time by his transfer to another hospital for the fitting of an artificial eye.

Streptomycin therapy was terminated on September 3. The gum lesion had completely healed by August 15. The bacteria were 100 per cent resistant to 1 microgram of streptomycin and no growth in 10 micrograms. At discharge, on Feb. 20, 1950, the patient's sputum had been negative for tubercle bacilli and his oral lesion had remained healed.

Comment.—It is believed that a tuberculous lesion of this area did not exist at the time of the tooth extraction and that the infection developed either postoperatively or in an area of inflammation produced by irritation from the lower denture.

Case 5.—E. M., a 33 year old patient, was admitted for far advanced active bilateral pulmonary tuberculosis of the reinfection type, with severe symptoms. His sputum was positive for tubercle bacilli.

Examination showed a draining sinus on the left cheek penetrating the skin and the mucosa and apparently originating from the mandibular region. The patient stated that this condition had existed for about one year and that he had been told it might have begun in the area of an abscessed lower left first molar. A roentgenogram of the mandible showed no abnormalities. In the drainage from the fistula tubercle bacilli could be demonstrated by culture.

The patient also suffered from tuberculous laryngitis. An ulcer of the left arytenoid region was seen, with impaired motility of the left vocal cord.

Originally 1.0 Gm. of streptomycin was given for one week; then the regimen was changed to dihydrostreptomycin, 3.0 Gm. daily, because tuberculous meningitis developed. Tubercle bacilli were isolated from the spinal fluid on March 31, 1949.

On May 10 the sinus on the left cheek had stopped draining. The fistula remained dry and was closing by granulation tissue. The larynx had greatly improved.

The resistance to the drug changed within six weeks from 10 per cent of the bacteria resistant to 1 microgram to 100 per cent resistant to 1 microgram, 50 per cent resistant to 10 micrograms and 25 per cent resistant to 100 micrograms.

The patient had refused intrathecal therapy and died of tuberculous meningitis on June 24.

Comment.—This is the only patient in our series who did not survive. He died of tuberculous meningitis.

Case 6.—S. C., a 54 year old white man, was admitted here with a diagnosis of far advanced active pulmonary tuberculosis of the reinfection type with moderate symptoms. He had bilateral fibroulcerative lesions in the upper lobes. The sputum was positive for tubercle bacilli.

During routine examination on April 21, 1949 granulomatous tissue on the mucosa of the gum and adjoining floor of the mouth, around the area of the right lower first molar, was observed. The patient was unaware of the presence of the lesion in this area. A biopsy was performed, and a diagnosis of tuberculosis was reported on April 29.

On May 4 administration of 1.0 Gm. of streptomycin intramuscularly daily was begun and its use continued for 42 days, until June 14. On June 1, the diseased area of the mouth was greatly improved, and on June 14 the lesion of the oral mucosa was completely healed. The patient left the hospital against medical advice on June 15.

Comment.—This case represents one of the instances in which the patient was unaware of the existence of an oral lesion. The diagnosis was made during routine examination and proved by biopsy.

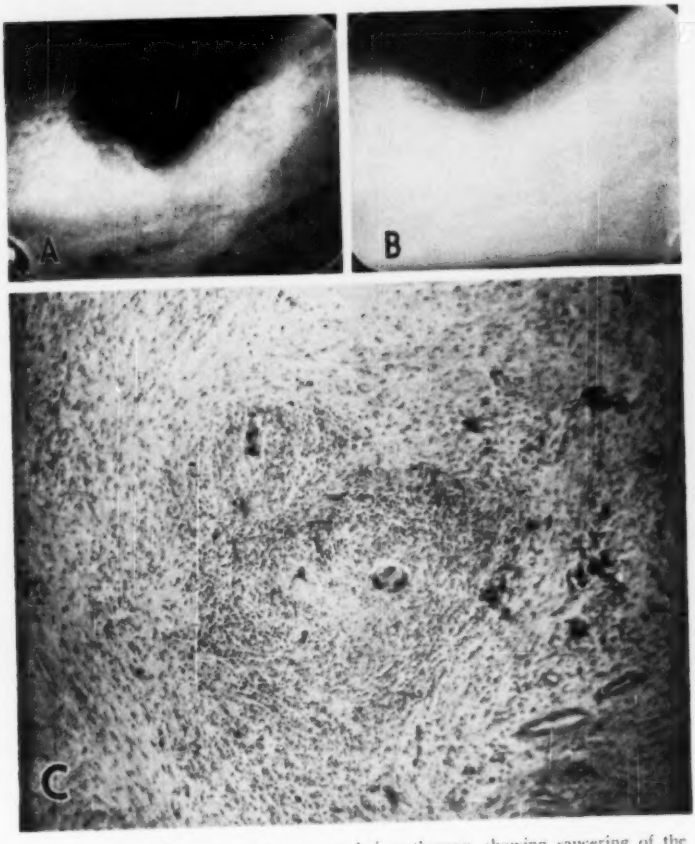

Fig. 1 (case 7).—*A*, roentgenogram before therapy, showing saucering of the bone from the crest of the ridge extending about one third through the mandible. *B*, roentgenogram six months later, showing healing of the lesion. *C*, photomicrograph of a section of the submucosa in the area of the molar, showing conglomerate tubercles.

CASE 7.—R. E. G., a 41 year old white man, entered this hospital with moderately advanced bilateral pulmonary tuberculosis of the reinfection type. Cavities were present on the right side. His sputum was positive for tubercle bacilli.

The patient stated that about two years previously the only remaining tooth on the right lower posterior jaw had become loose. The patient had removed

it himself. He had had no complaints after the removal of this tooth. On Nov. 7, 1949, during routine examination in preparation for making dentures, pus was seen exuding from a fistula near the crest of the ridge. Roentgenograms showed saucering of the bone from the crest of the ridge extending about one third through the mandible. At the crest of the ridge the lesion was about 15 mm. long. The impression was residual radicular cyst or tuberculosis lesion (fig. 1 *A*).

On November 11, the area of the second and third lower right molars was opened surgically. A fairly firm mass resembling a granuloma was encountered. The lesion separated easily from the surrounding bone, and a complete removal was attempted. The underlying bone appeared smooth and well formed. The report on the biopsy from the soft tissue of the area of the second and third lower right molars stated that the buccal mucous membrane, capped by a thin layer of

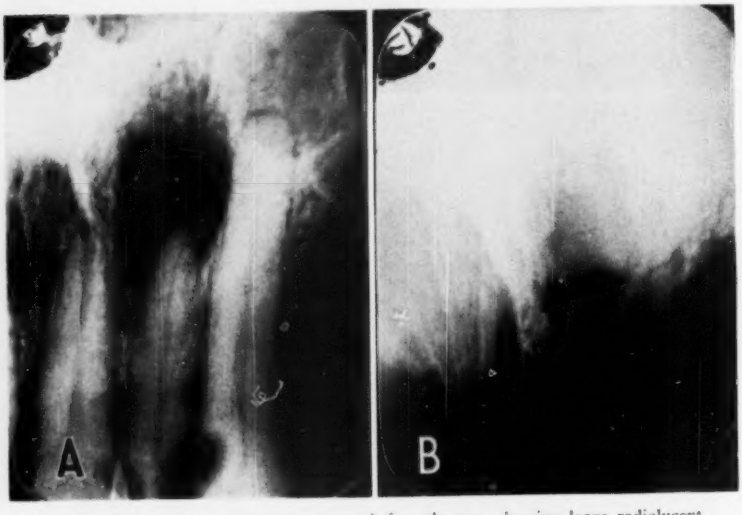

Fig. 2 (case 8).—*A*, roentgenogram before therapy, showing large radiolucent oral area, 10 mm. in diameter, about the apex of the tooth. *B*, roentgenogram five months after therapy, showing the concave area filled with tissue.

keratin, contained in the submucosa isolated and conglomerate tubercles. The impression was that the condition was tuberculosis (fig. 1 *C*).

Administration of 1.0 Gm. of dihydrostreptomycin intramuscularly and 12.0 Gm. of paraaminosalicylic acid orally was started on Dec. 1, 1949 and continued daily through Jan. 27, 1950, for 60 days.

The bacteria were sensitive to 1 microgram of dihydrostreptomycin on Nov. 18, 1949. After 60 days of dihydrostreptomcyin therapy, the bacilli were 50 per cent resistant to 1 microgram. No growth was observed in 10 micrograms.

Subsequent observations showed progressive healing of the lesion, and by Dec. 12, 1949 the area was practically filled in with normal tissue. By December 31 the area was completely healed. Later observations showed no recurrence of the lesion (fig. 1 *B*).

The patient did not gain any weight, and the condition of his lungs was unchanged.

Comment.—The area described apparently became infected with tubercle bacilli from the sputum at the time that the patient removed his own tooth.

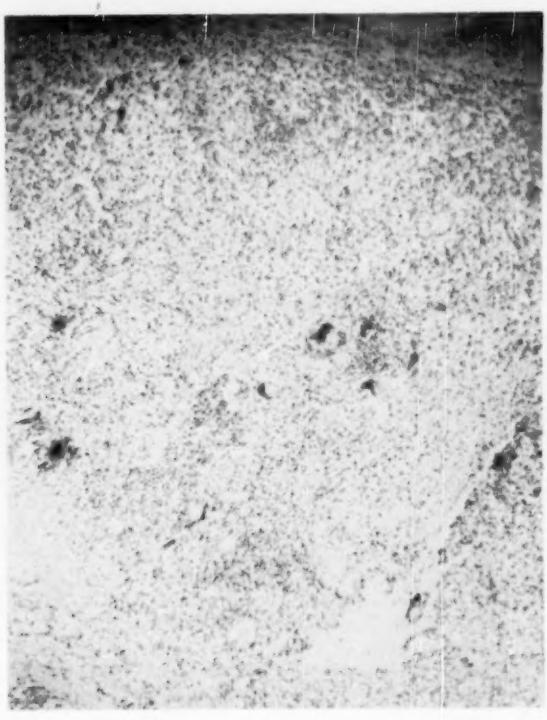

Fig. 3 (case 8).—Histological section of the lesion, showing tuberculous tissue.

Case 8.—R. W. H., a 33 year old white man, was admitted for far advanced pulmonary tuberculosis of the reinfection type, with cavitations in the upper lobe of the right lung. His sputum was positive for tubercle bacilli.

On routine dental examination on Nov. 15, 1949 scar tissue apical to the right upper lateral incisor was noted, in an area where apparently a fistula had previously opened into the buccal fold. The patient had no complaints and was unaware of his condition.

Roentgenograms showed a large radiolucent oval area, 10 mm. in diameter, about the apex of the tooth (fig. 2*A*). This tooth showed large cavities, both mesial and distal, with pulp communication. The bone margin was smooth and well defined. Results of vitality tests were negative.

On November 15, the upper right lateral incisor with the lesion and the scar tissue of the fistulous tract was removed. Grossly, the central portion of the lesion was jelly-like, but the peripheral portion was granulomatous. Histological examination revealed tuberculosis (fig. 3).

Administration daily of 1.0 Gm. of dihydrostreptomycin intramuscularly and 12.0 Gm. of paraaminosalicylic acid orally was started on December 12 and ended on April 3, 1950, after 120 days. Healing was progressing uneventfully, and by Jan. 5, 1950 the area was completely cured. On April 6 the concave area was filled with normal tissue (fig. 2 *B*).

On the sixtieth day of dihydrostreptomycin therapy 50 per cent of the bacteria were resistant to 1 microgram, and there was no growth in 10 micrograms of the drug.

Comment.—It is believed that a dental granuloma became infected with tuberculosis from sputum entering through the pulp and root canal of the tooth. The patient had some vomiting from paraaminosalicylic acid, which subsided when the drug was withheld for a few days.

Case 9.—J. J., a 54 year old patient, entered the hospital with far advanced pulmonary tuberculosis of the reinfection type. Bilateral disease with large cavitation in the upper lobe of the right lung was present. The sputum was positive for tubercle bacilli.

On routine dental examination on Feb. 15, 1950, a large radiolucent area, about 10 mm. in diameter, was noted around the apex of the lower left third molar, extending mesially to the alveolar crest. The surrounding margin of bone was smooth and well defined. The tooth was not sensitive, and there were only very small occlusal cavities. The tooth was loose; the surrounding tissue did not show any unusual changes clinically. The patient did not know previously that this condition existed. The rate of the growth was unknown but apparently was very slow.

On February 27 the tooth was extracted and a biopsy specimen taken from the surrounding area. Grossly the lesion appeared soft and jelly-like, except for the peripheral margin, which was somewhat firmer and granulomatous. Histological examination revealed several granulomas, each consisting of giant cells, epithelioid cells and lymphocytes, surrounded by a common capsule. The diagnosis was tuberculosis.

On March 25 daily administration of 1.0 Gm. of dihydrostreptomycin intramuscularly and 12.0 Gm. of paraaminosalicylic acid orally was initiated, and the therapy was continued daily for 120 days.

On April 6 the area of the third left molar was partially covered with normal granulation tissue. By May 16 continued progress in healing had been made. On May 23 the lesion had cured completely.

Comment.—The lesion began with tubercle bacilli from the sputum entering through the cervical margin. The infection originated in the peridental membrane. The bone loss was not due to infection within the bone but was caused by absorption from hyperemia within the adjacent tissues.

Case 10.—P. A. B., a 45 year old white man, was admitted to this hospital on March 28, 1950. Bilateral, active, far advanced pulmonary tuberculosis of the reinfection type was diagnosed. Caseocavernous lesions were present in the upper lobe of the right lung. The patient was extremely emaciated and complained of a fistula in ano. His sputum was positive for tubercle bacilli.

The patient stated that around Christmas 1949 a sore had developed in the left corner of his mouth which at first formed a scab and then became an open sore, showing no tendency to heal. The area of the sore had increased since the onset and had extended into the skin and the mucous membrane at the corner of the mouth. In January 1950 the patient began to complain of hoarseness and speech difficulties. The condition grew worse progressively until the time of his admission, when swallowing had become very painful and strangling, even on liquids, resulted.

Examination showed an ulcerating lesion in the left corner of the mouth, invading the lower lip and extending into the skin and buccal membrane, where there existed a granulomatous area (fig. 4*A*). The entire surface bled easily on touch. Examination of the larynx revealed ulcerative destruction of the upper margin of the epiglottis, with granulomatous thickening of the interarytenoid area.

Fig. 4 (case 10).—*A*, lesion before therapy, showing ulcerating lesion in the left corner of the mouth invading the lower lip and extending into the skin and buccal membrane. *B*, lesion after four weeks of therapy, showing the skin around the left corner of the lip healed completely.

Biopsies of the lesions of the lip were performed. Microscopically numerous giant cells were seen, few of which were mature and most of which were rather young with a few to several nuclei. Epithelioid cells were present in abundance. In several fields their arrangement was in tubercles. Fibrosis, pus cells and capillaries were abundant. The impression was tuberculosis (fig. 5).

On March 29 daily administration of 1.0 Gm. of dihydrostreptomycin intramuscularly and 12.0 Gm. of paraaminosalicylic acid orally was begun, and the therapy was continued for 120 days.

On April 26 the area of the lip had nearly completely healed. There was a radiating cicatrix forming on the buccal surface and the skin of the left angle of the lip. The larynx was greatly improved. The patient had been able to eat well and had gained weight.

On May 3 the skin around the left corner of the lip had healed completely. The buccal lesion showed a healthy but uneven surface (fig. 4 *B*). The ulcer of the epiglottis had disappeared and been replaced by healthy granulation tissue. The interarytenoid area was normal. The lip and the larynx have remained healed from that time on.

Comment.—Tuberculosis involving the lip is very rare. This case represents one of the earliest in which the condition was treated with dihydrostreptomycin and paraaminosalicylic acid and may well constitute the first reported case in which this therapy was used.

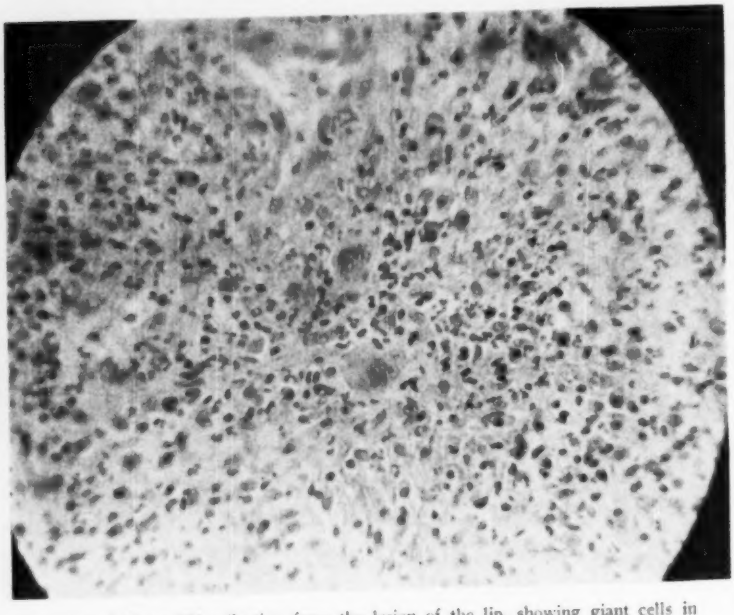

Fig. 5. (case 10).—Section from the lesion of the lip, showing giant cells in various stages of formation, epithelioid cells, fibrosis and capillary formation.

Case 11.—H. D., a 57 year old white patient, entered this hospital with a diagnosis of far advanced pulmonary tuberculosis of the reinfection type. His sputum was positive for tubercle bacilli.

On May 24, 1948 a biopsy of a specimen from a chronic ulceration of the mucosa at the lingual surface of the left mandible was performed. This area showed a somewhat deeper pink color surrounding a flat and fissured lesion. The patient had noticed some irritation in this area for about three weeks.

Microsection showed stratified squamous epithelium. Beneath the epithelium was a granulomatous inflammatory process with numerous lymphocytes and plasma cells. In one area multinucleated giant cells of the Langhans type were observed. The diagnosis was tuberculosis.

The patient had been treated with ultraviolet therapy without success. He left the hospital against medical advice on September 3, before streptomycin therapy could be started.

Comment.—This case, while not including use of streptomycin therapy, is reported here because it appears in our statistics (above) as a case of proved oral tuberculosis.

SUMMARY AND CONCLUSIONS

After a brief review of the essential literature, 11 cases of oral tuberculosis are reported. The buccal mucosa was involved in five patients and the tongue in another three. Two lesions occurred on the alveolar process, and one was located on the lower lip. Tuberculosis involving the fauces, the tonsils and the pharynx was intentionally omitted in this report and was mentioned only when occurring in addition to other oral pathologic conditions.

In all our cases the lesions developed in the presence of sputum positive for acid-fast bacilli due to pulmonary tuberculosis. However, a sputum positive for the bacilli alone was not considered sufficient evidence for establishing a diagnosis of tuberculosis for patients with suggestive oral lesions. In ten of our cases the diagnosis of tuberculosis was verified by biopsy. In one instance tubercle bacilli could be demonstrated in the culture from the secretion of a fistula.

Ten of the 11 patients were treated with streptomycin or dihydrostreptomycin and paraaminosalicylic acid according to the randomization of the Veterans Administrations streptomycin protocol. All oral lesions healed from within 10 days to about two months under this regimen. Two recurrences were observed, and these lesions remained healed after a second course of streptomycin therapy.

Our series is too small and the time elapsed, by necessity, must be insufficient for a final evaluation of the results obtained—after all, streptomycin was introduced clinically only about five years ago. But it may be stated safely that treatment with streptomycin or dihydrostreptomycin represents a simple and effective method of combating oral tuberculosis. Additional therapeutic procedures may only rarely become necessary. The rate of emergence of streptomycin-resistant tubercle bacilli assumed a well known pattern, similar to that described in the reports of other investigators.

TYMPANOMEATAL MEMBRANE IN THE FENESTRATION OPERATION

Its Relation to Bony Closure of the Fenestra

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NEW YORK

THE EVOLUTION of otosclerosis surgery in America received its first impetus in 1937, when Sourdille,[1] professor of otolaryngology at the University of Strasbourg, described his fenestration operation at the New York Academy of Medicine. Sourdille's work was inspired by watching Holmgren operate for otosclerosis in 1924. Attempts at fenestration of the labyrinth for otosclerotic deafness had been made by Passow[2] in 1896, Jenkins[3] in 1912 and Bárány[4] in 1914. Holmgren[5] in 1916 proved that the membranous labyrinth could be exposed without endangering the cochlea and that improvement in hearing could be achieved in properly selected cases of deafness due to otosclerosis. Lempert[6] in 1938 made a monumental contribution when he described the one stage fenestration operation. Osteogenetic closure of the operative fenestra has always been and still is the chief obstacle to the success of the fenetration. Recently, the contributions of Lempert and associates,[7] G. E. Shambaugh Jr. and Juers,[8] E. P. Fowler Jr.,[9] Lindsay[10]

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7. (*a*) Lempert, J.: Fenestra Nov-Ovalis: A New Oval Window for the Improvement of Hearing in Cases of Otosclerosis, Arch. Otolaryng. **34**:880 (Nov.) 1941; (*b*) Lempert Fenestra Nov-Ovalis with Mobile Stopple: New Advance in Surgical Treatment for Clinical Otosclerosis Evolved as Result of

and others have gone a long way to reduce the number of such fenestral closures, yet much more remains to be discovered to solve this difficult problem.

The purpose of this paper is to call attention to a surgical cause for osteogenetic fenestral closure with consequent failure in hearing improvement. Such closure is due to undiscovered fractured splinters of bone attached to the tympanomeatal flap precisely where it covers the fenestra. The portion of the tympanomeatal flap which covers the fenestra must be absolutely free from attached splinters of fractured bone, because such bone fragments, when present, exert a powerful stimulus to osteogenesis, thereby closing the fenestra. These bits of fractured bone which adhere to the flap result from removal of the underlying bony walls of the auditory canal. Shambaugh and Juers demonstrated the osteogenetic effect of bone dust and bone particles in monkeys with fenestration. Lempert corroborated their work.

In discussing this problem of bone spicules on the periosteal surface of the flap, Lempert [7b] in 1945 made the following statement:

> The inner surface of the tympanomeatal membrane, which is destined to cover the region of the fenestra, was always carefully examined with the Zeiss magnifying glasses in a search for minute and extremely thin splinters of bone, which were commonly found to be attached and adherent to it as a result of the removal of the bony walls of the external auditory canal. Such splinters when observed were removed, care being taken not to perforate the tympanomeatal membrane. Splinters of bone when unobserved and not removed will rapidly cause closure of the new fenestra by bone regeneration—much more rapidly than when the regeneration of bone originates solely from the freshly cut bony walls of the fenestra.

Shambaugh and Juers [8b] in 1946 stated:

> If the periosteal surface of the skin flap is minutely searched with the binocular loupe for embedded and adherent bone particles, a surprising number of such particles can be discovered and removed with fine forceps, a small periosteal elevator and a manicure scissors.

Research Study of 1000 Cases in which Fenestration Has Been Performed During Last 7 Years, ibid. **41**:1 (Jan.) 1945; (*c*) Bone-Dust-Free Lempert Fenestra Nov-Ovalis: New Evolutionary Development of Surgical Treatment of Clinical Otosclerosis, ibid. **47**:280 (March) 1948. (*d*) Lempert, J.; Meltzer, P. E.; Schall, L. A., and Wolff, D.: Osteogenesis Following Fenestration of the Vestibular Labyrinth of the Rhesus Monkey: Controlled Experimental Study, ibid. **46**:512 (Oct.) 1947. (*e*) Footnote 8.

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Unless specifically searched for under magnified vision, these bone scales and particles will escape detection.

With this problem in mind, the fenestration operation was performed on 100 fresh adult cadavers, and at the conclusion of each operation the intact and entire tympanomeatal flap was removed. The technic of creating the tympanomeatal flap was the standard one described by Lempert. Each stretched-out flap was then searched for bone particles with a binocular loupe and a tenfold magnification loupe. After all detected bone particles were removed, a roentgenogram was made of the flaps. The roentgenogram revealed a surprising number of random

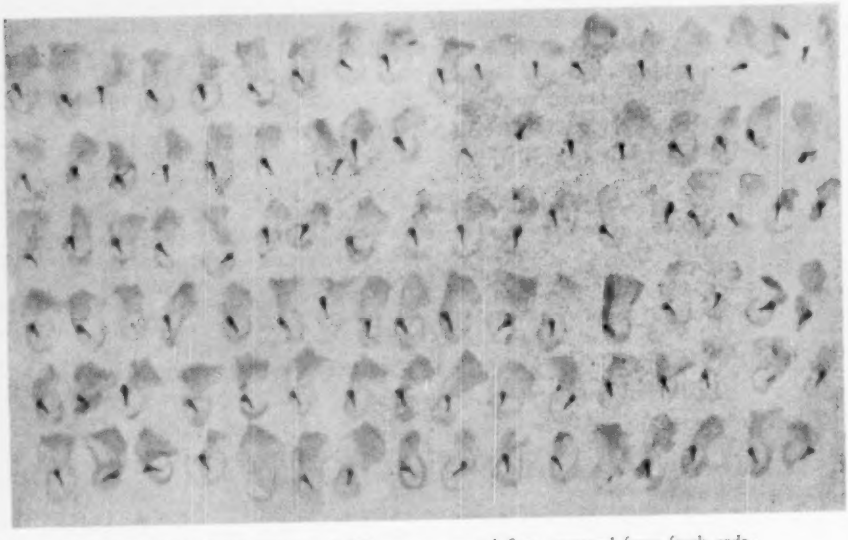

Fig. 1.—Roentgenogram of 100 tympanomeatal flaps removed from fresh cadavers after the fenestration operation was completed. The inner aspect of these flaps were searched for adherent bone particles with tenfold magnification, and all visible particles of bone were removed. A roentgenogram was made of the specimens, and undetected bone particles were revealed in about half. Any bone particle adherent to that portion of the tympanomeatal flap which is destined to cover the fenestra may cause quick closure of the fenestra and consequent poor hearing. The handle of the malleus (head and neck removed during the operation) can be seen attached to the inner surface of the drum membrane.

bone particles and bone scales which had escaped detection in about half of the specimens (fig. 1). In other words, after careful removal of embedded bone particles from the periosteal surface of the tympanomeatal flap under tenfold magnification, there still remained many bone particles which could not be removed because they could not be detected. When

such undetected bone particles happen to make contact with the fenestra, osteogenesis and rapid closure of the new window with poor hearing are the result.

In order to discover the reasons for these observations, it became necessary to analyze the steps in the technic of creating the tympanomeatal flap. Lempert,[6] in 1938, in describing the resection of the bony walls of the canal to liberate the skin portion of the flap, said:

A specially devised narrow periosteal elevator is inserted between the cutaneous lining and the skeletonized posterior and superoposterior bony walls of the canal, and the cutaneous lining is gently separated from its attachment to the bony surface, 1 mm. at a time. Each millimeter of bony wall thus exposed is successively removed with a specially devised rongeur, up to the level of the bony capsule of the vertical portion of the facial nerve. The skeletonized superior and anterosuperior walls are removed up to the sulcus tympanicus by the same technic.

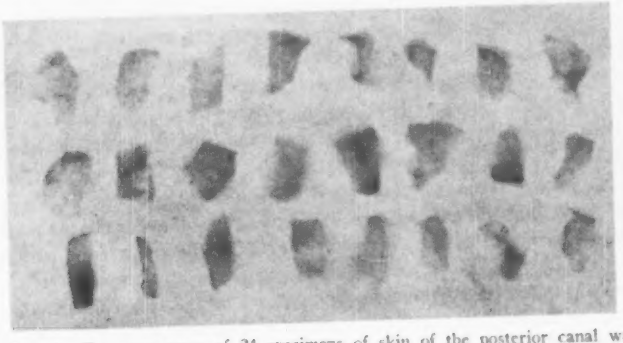

Fig. 2.—Roentgenogram of 24 specimens of skin of the posterior canal wall easily separated from the underlying bone at the onset of the fenestration operation before any bone work is begun. This separation insures absence of any bone particles on that portion of the tympanomeatal flap which always covers the fenestra. The roentgenogram reveals complete absence of any bone particles adherent to the flap which covers the fenestra. Osteogenetic closure of the fenestra from bone adherent to the flap is thus eliminated.

This very millimeter by millimeter method of bone removal frequently causes minute, unpredictable comminuted fractures just beyond the last millimeter of removed bone. These bone particles adhere to the inner surface of the flap and when in contact with the fenestra will cause its quick closure.

In order to obviate the bone spicules from adhering to the portion of the inner surface of the flap which is destined to cover the fenestra, I separated at once the skin from the posterior bony canal wall from the level of the mastoid cortex externally to the cavity of the middle ear internally before beginning any bone work. This skin peels away easily from the underlying bone. For this purpose a dull periosteal elevator

or, better, an ordinary manicure orange stick with rounded edges was used. The comparatively dull and soft orange stick was used to avoid scraping off thin scales from the osteogenetically active cambium layer of the underlying bone onto the periosteal surface of the flap. This area of skin of the posterior canal wall is limited above by adherent fibrous tissue that runs into the tympanosquamous suture and below by adherent fibrous tissue that enters the tympanomastoid suture. This area of skin of the posterior canal wall between the upper and lower fibrous attachments always covers the fenestra. Twenty-four of these specimens of meatal skin destined to cover the fenestra were resected and a roentgenogram made. All were found to be entirely free from any bone particles (*fig. 2*). The presence of bone particles adherent to other distant parts of the meatal portion of the flap would be immaterial, for those portions of the flap do not cover the fenestra. Since the meatal skin which covers the fenestra is entirely separated from the bone before the bony canal wall is bitten away, no fractured bone particles can adhere to this portion of the flap. It would seem, therefore, that Lempert's method of freeing the meatal skin from the bone of the posterior bony canal millimeter by millimeter should be modified in the manner here described to avoid closure of the fenestra by undetected fragments of bone adherent to the flap.

SUMMARY

When Lempert's technic is used, particles of bone adhere to the periosteal surface of the tympanomeatal flap. These particles are not all detectable under tenfold magnification.

Bony particles adherent to that portion of the tympanomeatal flap which is destined to cover the fenestra may cause osteogenetic closure.

A modification of Lempert's technic is described in which that portion of the tympanomeatal flap which always covers the fenestra will remain completely free from any adherent fractured pieces of bone, thereby eliminating an avoidable cause for failure of the fenestration operation.

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USE OF VINEGAR AS AN ANTIBIOTIC IN THE TREATMENT OF CHRONIC MIDDLE EAR DISEASE

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THE ADVENT of the sulfonamides and antibiotics provided physicians with means of combating most of the infections met in their daily practice. The spectrums of effectiveness of these drugs cover most of the common organisms, and the diseases these organisms produce usually wilt with the action of these drugs. However, there still remains, in significant numbers, a group of chronic infections—a residue of the presulfonamide period or the result of recent infection—with a fast or resistant organism. In this group is chronic purulent middle ear disease. Patients usually have a profuse, foul, mucopurulent discharge due to infection from a mixture of organisms. These infections have persisted despite all medical treatment and, frequently, rigorous repeated surgical treatment. One reason for this situation is that many of the organisms are in the family of Pseudomonas aeruginosa and Proteus vulgaris, which up to now have defied treatment. A second reason is that these patients may have associated bone necrosis or cholesteatoma acting as foreign bodies or foci of reinfection.

An earlier paper[1] reports the treatment of external otitis with the use of a wet dressing of acetic acid and aluminum acetate (Burow's solution). Household vinegar was used as a source of acetic acid. Often, the vinegar was used directly, without dilution, and it was effective in clearing the infection in the skin of the external auditory canal. Vinegar was impressive and dramatic in its antibacterial action and completely innocuous to the tissue. These results led to the use of vinegar in chronic middle ear infections.

The use of vinegar in medicine dates back to antiquity. Beyond doubt, vinegar is the first antibiotic known to man, a bacterial product antagonistic to other organisms. Thus, the principle of antibiosis actually has been used by physicians since the earliest civilizations. Topical use of vinegar in the ear is described in an Assyrian medical

1. Ochs, I. L.: Treatment of External Otitis: A Simple and Effective Technic, J. A. M. A. **142**:1361 (April 29) 1950.

text, translated by Thompson.[2] The Jewish Encyclopedia mentions several medical uses of vinegar in biblical times, including its use as a wet dressing on wounds. The Encyclopedia Britannica states that vinegar was used medically by Hippocrates in 400 B. C. The new Oxford English Dictionary quotes Holland in 1601 as follows: "Vinegre dregs are knowne to be verie good for to heale burns."

It is often used today as a wet dressing in treating Ps. aeruginosa infections of the skin and as a vaginal douche. Despite the extensive and ancient use, references in the literature to such use are few. Basic studies on the antibacterial action of acetic acid have been made, and these are impressive. The most recent of these studies is described in a paper by Owen[3] on the use of acetic acid as an inhibitor of gram-negative bacilli in culture media. Acetic acid was used as a means of suppressing the growth of gram-negative organisms in cultures from mixed infections. The gram-negative bacilli did not overgrow the culture media and confuse the bacteriologist in her attempt to find the true pathogen. She found that by adding 0.1 cc. of a 10 per cent acetic acid solution to 15 cc. of beef heart infusion broth, a solution of p_H 6.0 to 5.5 resulted. This mixture inhibited the growth of gram-negative organisms. One-half cubic centimeter of 10 per cent acetic acid solution added to the same culture resulted in a solution of p_H 4.5. This mixture, which approximated one third of 1 per cent acid, completely inhibited growth of all organisms. Owen made the following statement. "A few experiments were done using hydrochloric and sulphuric acid in parallel concentrations with acetic acid, but they were entirely unsuccessful."

Levine and Fellers[4] reported a similar experiment in 1940 with about the same results, especially as to the bactericidal powers of acetic acid as compared to other acids. A third study in this field was done by Taylor[5] in 1916 in treating wound infections in World War I. He found that butyric, propionic, malic, tartaric, and acetic acids were effective in inhibiting the growth of Ps. aeruginosa. Hydrochloric acid and nitric acid were not effective. With the use of a wet dressing of a 1 per cent solution of acetic acid, no wounds showed Ps. aeruginosa infection after one week, while other dressings, including those with modified Dakin's solution (diluted sodium hypochlorite solution) and

2. Thompson, R. C.: Assyrian Prescriptions for Diseases of the Ears, J. Roy. Asiatic Soc., 1931.

3. Owen, C. R.: Acetic Acid Inhibition of Gram-Negative Bacilli in Culture Media, J. Bact. **52**:353, 1946.

4. Levine, A. S., and Fellers, C. R.: Inhibiting Effect of Acetic Acid upon Microorganisms in Presence of Sodium Chloride and Sucrose, J. Bact. **40**:255, 1940.

5. Taylor, K.: Treatment of Pyocyaneus Infections, J. A. M. A. **67**:1598 (Nov. 25) 1916.

saponated cresol solution, showed 60 per cent infection after the second week and 37 per cent after the third week.

Several European investigators have done basic work and have shown the bactericidal effect of acetic acid on other organisms. Meyer,[6] in 1937, showed that acetic acid was amebicidal, and stated that it "may have therapeutic value." No clinical follow-up observations were reported in the literature. Several authors have demonstrated the effect of acetic acid on the tubercle bacillus. This organism is relatively resistant, but acetic acid is shown to be considerably more lethal than other acids.

Saenz and associates[7] stated that a 5 per cent solution of acetic acid killed these organisms in a half-hour. The organisms tolerated a 10 per cent solution of sulfuric acid for an indeterminate period. Pulcher[8] reported similar findings on the relative effectiveness of acetic acid and sulfuric acid on tubercle bacillus, using culture on Petroff medium and guinea pig inoculation. He found that a 32 per cent sulfuric acid solution failed to kill the organism after five and one-half hours. Six per cent acetic acid solution after five hours sterilized suspensions of the bacteria, as determined by culture and guinea pig inoculation. Two other European authors[9] showed the practical value of the disinfecting power of acetic acid in the treatment of salad greens. Van den Branden and Geens found that lettuce leaves contaminated with the organisms of night-soil were sterilized after five minutes in a 6 per cent solution of acetic acid. Perini did the same experiment, except that the lettuce leaves were first dipped in the cultures of common intestinal organisms of the colon and in typhoid, paratyphoid and cholera organisms. The leaves were sterilized after ten minutes in his vinegar solution.

The papers mentioned above describe the effectiveness of acetic acid against a wide range of organisms in vitro. It was shown by these experiments that acetic acid in dilution as low as one-third per cent is capable of inhibiting not only the gram-negative organism but also many of the gram-positive pathogens.

In addition to the work of Taylor,[5] there are several other clinical studies on the effectiveness of acetic acid wet dressings in treating

6. Meyer, W.: Amoebecidal Action of Organic Acids from Biologic Sources, Zentralbl. f. Bakt. (Abt. 1.) **139**:382 (Aug. 10) 1937.

7. Saenz, A.: Comparative Toxicity of Solutions of Sulfuric and Acetic Acid on Tubercle Bacillus, Compt. rend. soc. de biol. **117**:1072, 1934.

8. Pulcher, C.: Effects of Sulphuric and Acetic Acid on Tubercle Bacillus, Arch. per le sc. med. **50**:145, 1927.

9. Van den Branden, F., and Geens, J.: Attempts at Destruction of Bacteria on Lettuce with Vinegar, Ann. Soc. belge de méd. trop. **18**:297, 1938. Perini, P. E.: Bactericidal Power of Vinegar Treated with Catadyn, Ann. d'ig. **46**:541, 1936.

infected wounds and skin. Rand[10] recommended the use of a 2 per cent solution of acetic acid or household vinegar to clear granulations of P's. aeruginosa infection before attempting skin graft.

Ludwig,[11] while working in a naval hospital in the Pacific area during the war, treated 358 patients with burns with saline glycerin solution containing one-half per cent acetic acid. He found that "the foul odor usually associated with burns disappeared in one to two days"; that no maceration followed its use; that a minimum of grafting was necessary under this regimen, and that epithelization appeared to be faster than with other methods. He came to the following conclusion: "These dressings clean up the burns very rapidly, resulting in clean granulation surfaces suitable for early skin graft." In another article, Ludwig stated that he also used the same solution as a wet dressing over skin grafts. In 28 cases, a wet dressing of one-half per cent acetic acid solution was used to secure the graft to the wound and to control infection. He concluded that this procedure permitted more rapid healing because of the absence of infection.

In the series of patients reported on in this paper, household vinegar, which is the equivalent of 5 per cent acetic acid, was used to treat 38 patients with chronic middle ear infection. In 30 cases it succeeded in eliminating the infection quickly and effectively. The action of this treatment is rapid and dramatic; there is no question of its efficacy.

METHOD

The ears are painstakingly cleaned of all discharge by mopping with cotton applicators. The use of a fine suction tube is often necessary when the exudate is particularly tenacious. After a thorough cleaning of the ear, hydrogen peroxide is instilled to help in the removal of any residual debris. The ear is then dried as completely as possible. The patient is placed with his head down and the affected ear upward. Boiled household vinegar is instilled into the canal to fill it. To help the vinegar penetrate into all the cavities of the ear, suction is applied with a snugly fitting bulb syringe in the auditory canal. This procedure, in effect, displaces the air from the cavities of the ear and mastoid and permits the vinegar to reach these ordinarily inaccessible cells. A firmly fitting cotton tampon is then placed in the auditory canal against the drum membrane, and more vinegar is added to saturate the tampon thoroughly. The patient returns after two days. The tampon is removed, and, if any exudate is present, the procedure is repeated. The

10. Rand, B. K.: Use of the Thiersch Skin Graft, Brit. M. J. **1**:846, 1940.

11. Ludwig, F. E.: Use of Saline Solution, Glycerine and Acetic Acid in Care of Burns: Odorless Method of Treating Burns, Surgery **19**:486, 1946: Use of Acetic Acid-Glycerin-Saline Solution in Skin Grafting, ibid. **19**:492, 1946.

patient with chronic ear disease does not experience pain with this technic beyond a minor sting. It should be mentioned that in the treatment of one ear with an acute condition the patient evinced considerable pain; however, the treatment was successful. When the middle ear is found to be free from discharge, an attempt is made to close the perforation by cauterization of its margins with the use of a silver nitrate bead.

RESULTS

The procedure often results in rapid and complete elimination of the chronic infection, frequently after one treatment. In the past two years, 30 patients with 36 infected ears have been successfully treated. None of these ears required more than three instillations of vinegar to eliminate the infection. Most of the patients had been through the gamut of sulfonamides and antibiotics, without any effect whatsoever on the course of the disease.

In addition, there were eight patients who failed to respond to the treatment. The bacteria cultured in the cases of therapeutic failure included hemolytic Streptococcus, hemolytic Staphylococcus, Staph. aureus, Proteus and diphtheroid. The failure in these cases was probably due to mechanical problems, the infection being too deep-seated to be reached by the solution. Of the 36 infected ears, all are dry at the present writing except one. In five, recurrences developed with an acute infection of the upper respiratory tract. In four, the recurrences responded promptly to one application of the same treatment. The condition in a 68 year old patient, who had had a discharging ear since childhood, cleared promptly in July 1948 and the ear remained free from discharge until December 1949, when a recurrence developed with an acute infection of the upper respiratory tract. At this time, in addition to the Proteus, nonhemolytic Streptococcus and diphtheroid were cultured. The condition in this patient resisted all local and systemic medication, and she still has a persistent, foul discharge from the ear.

Following are summaries of representative cases. The first four constitute the total number of patients with chronic middle ear infections who were referred for treatment by the Veterans Administration. Their histories and findings are a matter of Government service record.

REPORT OF REPRESENTATIVE CASES

M. L., a 24 year old white woman, had a bilateral purulent disease of the middle ear dating from 1945, when she was in the Navy. Despite vigorous treatment with sulfonamides and penicillin, her ears failed to clear and continued to drain foul, purulent material. Both ears cleared after two treatments with vinegar. In addition, the perforations in both ears were closed with the use of silver nitrate cautery. Her left ear has remained dry to date, a period of two years. There was

a recurrence in her right ear after an acute infection of the upper respiratory tract four months after treatment. This recurrence cleared after one treatment, and, at the present time, both ears are dry with intact drum membranes.

G. B. D., a 28 year old white man, had a middle ear disease after an acute infection of the upper respiratory tract while in the Army in 1943, where he was treated with sulfonamides. Culture of the discharge produced Staph. aureus, diphtheroids and Hemophilus influenzae. The persistent, foul and profuse purulent discharge continued, with loss of hearing, until he received the vinegar displacement treatment. After one application the discharge stopped and the ear remained dry. This perforation was successfully closed by cauterization of the margin. There has been no recurrence to date, a period of fourteen months.

V. A. H., a 28 year old white man, had a middle ear disease following an acute infection of the upper respiratory tract while in the Army. He had an intermittent aural discharge of foul, purulent material. At the time he was seen, his ear had been discharging for six months without interruption. This ear became dry after three treatments. The perforation was closed with a silver nitrate cautery, and there has been no recurrence to date, a period of fourteen months.

L. A. N., a 28 year old white man, had intermittent draining of the right ear with loss of hearing since an initial bout of infection in 1944 while in the Navy. This ear had been discharging for about two months when treatment was started. A moderate amount of foul, purulent discharge was coming from a marginal perforation occupying the anterior one third of the drum. The discharge cleared after three vinegar treatments, and the ear has remained dry to date, a period of eighteen months. No attempt was made to close the perforation because the patient objected to the pain when the cautery was applied.

V. M., a 20 year old white man, had an uninterrupted draining condition of the middle ear since the age of 9. The drum membrane had a 2 mm. perforation in the upper anterior portion not involving the margin. A bud of granulations protruded through this perforation. Culture of the discharge was reported as growing Escherichia coli and P. mirabilis. This condition cleared after three displacement treatments plus cautery of the granulations with a silver nitrate bead. His ear has remained dry to date, a period of one year.

A. T., a 22 year old white man, had a bilateral purulent middle ear disease since infancy. When first seen, he had an acute mastoiditis on the left, with a temperature of 104 F., cellulitis over the mastoid and zygomatic area and spasticity of the neck muscles. Culture of the discharge grew Ps. aeruginosa and Staph. aureus. He was placed under treatment with penicillin and streptomycin, and a radical mastoidectomy was done, resulting in improvement of the symptoms, but the discharge persisted. Two months postoperatively, the discharge stopped after two treatments with the vinegar, and the ear has remained dry to date, a period of nineteen months. The right ear was treated with vinegar but failed to clear until a radical mastoidectomy was done.

E. C., a 25 year old white woman, had a foul, purulent aural discharge. At the time she was treated, she stated that she had had continuous, uninterrupted discharge for at least two years. Examination revealed a central perforation in the lower anterior part of the drum membrane and a marginal perforation in the upper posterior part of the membrane, each about 2 mm. in diameter. Through the lower perforation a pulsating purulent exudate drained, and through the upper, a bud of granulation protruded. The discharge in this ear stopped after two vinegar displacement treatments, and both perforations healed after cauterization of the margins and granulations with silver nitrate. There have been no recurrences to

date, a period of eight months, and the patient believes that her hearing is much improved. She is able to hear the telephone with the left ear, which she could not do before.

I. S., a 36 year old white woman, had had a fenestration operation in 1943 for otosclerosis and since then has had an aural discharge of varying degree. In the past year she was treated once a month, by removal of the discharge, which was foul and cheesy, together with a cast of the mastoid cavity and middle ear composed of slough from the keratin layer of the skin plus bacterial debris. After the ear was cleaned, sulfadiazine powder was dusted into the cavity. With this procedure, severe vertigo developed. The sulfadiazine powder had little deterrent effect on the organism. The skin covering the labyrinth was probably quite thin, and the possibility of damage to the membranous labyrinth or to its neural component by the absorption of the vinegar discouraged its use. However, in view of the failure of other remedies, and with the increasing success with the use of vinegar in the middle ears of other patients, it was applied cautiously, without suction, after a thorough cleansing. To date, a period of eight months, the ear has remained dry, and on inspection the skin lining the cavity of the middle ear and the mastoid is normally pale, thin and without exudate. It would appear from this picture that the production of keratin by the grafted skin in the mastoid cavity is dependent on irritation by an infecting organism.

COMMENT

The effectiveness of the procedure of vinegar displacement is obvious from the rapidity and completeness of its action in cases in which the ear had previously drained for years. It is possible that good results could be obtained with the use of a much more dilute solution of acetic acid. The addition of a selected detergent might be of some help in assisting the bactericidal effect because of its wetting action.

The acetate ion derived from a salt may be better tolerated by the body, and this basic problem must be investigated. Theodore's work [12] on the use of sodium propionate lends further weight to this possibility.

CONCLUSION

The vinegar displacement method is effective in clearing chronic infection in the middle ear.

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12. Theodore, F. H.: Use of Propionates in Ophthalmology: A Preliminary Report, Arch. Ophth. **41**:83 (Jan.) 1949.

Case Reports

RHABDOMYOSARCOMA OF THE MAXILLARY ANTRUM

Seven Year Survival Following Surgical Excision and Radiation Therapy

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Striated muscle tumors involving the nasal accessory sinuses apparently are extremely rare. Because of this it seems worth while to report a case of extensive rhabdomyosarcoma of the maxillary antrum. A survival period of seven years after treatment is especially gratifying.

Regarding tumors of striated muscle, as with many rare and unusual tumors, the literature seems confusing, especially in the matter of prognosis. Jönsson [1] considered rhabdomyosarcoma the most malignant and radioresistant of all the sarcomas of soft parts. Murphy, Dockerty and Broders [2] pointed out that myoblastomas, particularly the uniform type, are in general slowly growing and clinically benign. They presented evidence that the polymorphous myoblastomas are more malignant histologically than clinically but that in general they tend to shade into rhabdomyosarcomas.

Stout,[3] too, pointed out that there was a difference between granular cell myoblastoma and the genuine rhabdomyosarcoma. Thus discrepancies may be due to differences in opinion in the classification of the specific tumors reported. In this connection the classification of Stout seems sound and reliable.

Broder's method of grading tumors on the basis of 1 to 4 depending on the degree of malignancy regardless of the morphologic pattern and origin seems practical when applied to rhabdomyosarcoma. Tumors in the group grade 1 have a more or less slow course, while tumors of grade 4, in our experience, seem hopeless (Eisenberg,[4] Moore [5] Mandeville [6]). Karatay [7] recently reported a case

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1. Jönsson, G.: Malignant Tumors of the Skeletal Muscles, Fasciae, Joint Capsules, Tendon Sheaths and Serous Bursae, Acta radiol., 1938, supp. 36, pp. 1-304.

2. Murphy, G. H.; Dockerty, M. B., and Broders, A. C.: Myoblastoma, Am. J. Path. **25**:1157-1181 (Nov.) 1949.

3. Stout, A. P.: Rhabdomyosarcoma of the Skeletal Muscles, Ann. Surg. **123**:447-472 (March) 1946.

4. Eisenberg, S. J.: Rhabdomyosarcoma: Case Report with Response to Roentgen Therapy, Virginia M. Monthly **75**:27-28 (Jan.) 1948.

5. Moore, J. P.: Rhabdomyosarcoma: Its Dissemination and Response to Radiation, Virginia M. Monthly **76**:234-238 (May) 1949.

6. Mandeville, F. B.: Roentgen Ray Aspects of Sarcomas of Soft Tissues, J. South Carolina M. A. **39**:59-62 (March) 1943.

7. Karatay, S.: Rhabdomyosarcoma of the Middle Ear, Arch. Otolaryng. **50**: 330-334 (Sept.) 1949.

of rhabdomyosarcoma of the middle ear in a 3 year old Negro girl. The patient died at the age of 3½ years.

REPORT OF A CASE

A Negro aged 46 years, was first seen in the Ear, Nose and Throat Outpatient Clinic of the Medical College of Virginia on Feb. 8, 1943, because of headache and painful swelling of the right side of the upper jaw. Early in December 1942 he contracted a head cold, and one week later toothache developed in the right upper jaw. During the second week of January 1943 two right upper molar teeth were easily extracted, but within two days marked swelling developed in the right cheek, the right nostril became occluded and the associated facial pain was intense. When aspirin no longer relieved the pain, medical aid was sought.

Examination revealed a well developed Negro, moderately uncomfortable and with abnormal physical findings limited to the right facial, palatal and nasal structures. The lower right eyelid, the malar and facial soft tissues and the nasal membranes were edematous and prominent but did not suggest cellulitis. There was moderate pain on pressure over these areas. Inspection of the oral and buccal cavities showed poor dental hygiene and several scattered carious and infected teeth. The right side of the hard and soft palates and the alveolar soft tissues were edematous. The right upper molar region was free of teeth and replaced by a prominent granulomatous cauliflower-like mass bathed in pus. On palpation the mass bled freely. The right nares was completely obstructed by soft tissue edema of the middle and inferior turbinates and creamy yellow pus. These same findings were also noted in the right choana. A tentative clinical diagnosis of advanced neoplasm of the right maxillary antrum was favored over acute osteomyelitis of the maxilla, and hospitalization for study and treatment was advised.

Roentgenograms of the sinuses and the skull showed clouding of the right ethmoidal, frontal and maxillary sinuses and destruction of the anterior, lateral and superior bony walls of the right maxillary antrum. No pathologic changes were noted in the lungs. Determination of the blood hemoglobin, differential and total leukocyte counts and urinalysis gave normal values. The flocculation test for syphilis was reported to show a negative reaction.

Histologic study of tissue removed from the cauliflower-like mass on February 8 showed acute and chronic inflammation of granulomatous tissue with necrosis. Tissue specimens were removed a second time and were indicative of rhabdomyosarcoma. They were reviewed by several pathologists, and the findings will be considered in more detail in the comment which follows.

TREATMENT

On February 16, with the use of 2.5 per cent pentothal sodium solution intravenously and endotracheal anesthesia, the tumor was removed surgically as follows: An extended Caldwell-Luc type of mucous membrane incision was made from the upper midline to the region of the second molar tooth on the right. The anterolateral bony maxillary wall was found missing and replaced by a soft, whitish gray, tapioca-like, slippery, lumpy material which filled the antral space. The tumor tissue masses varied in size, some being less than 0.5 cm. in size and the largest mass measuring 3 by 2 by 1 cm. The tissue seemed under pressure and oozed from its confines. With the use of the index finger for palpation dissection, most of the tissue was readily removed and filled two 1 ounce (30 cc.) glasses. Some of it was lost by suction and on sponges. Grossly there seemed to be no well defined capsule as such. The floor of the orbit and the entire bony structure of the right antrum were absent except for the infraorbital ridge and the medial one third of the anterior floor of the hard palate.

The walls of the cavity were generally smooth throughout except in the region of the hard palate, which showed infected and granulomatous tissue and tumor presenting into the mouth. The soft tissues of the lateral wall were indurated and irregular. After all grossly visible abnormal tissue had been removed, including the right lateral two thirds of the hard palate remnants and the anterior half of the soft palate, 100 mg. of radium element in two 50 mg. capsules with 0.5 mm. platinum filter were placed in the space and maintained in place with gauze for ten hours for a total of one thousand milligram hours.

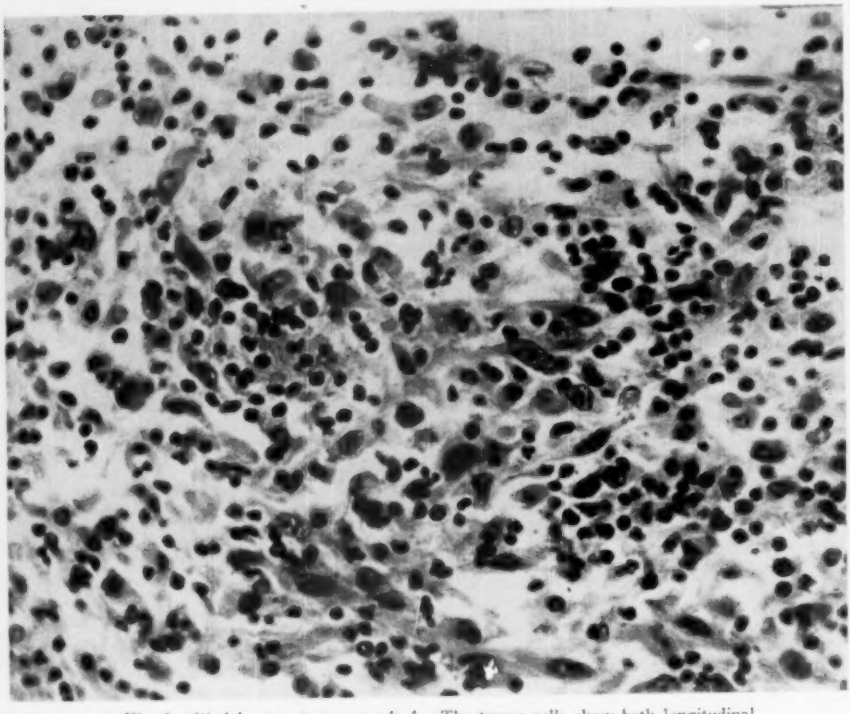

Fig. 1.—Rhabdomyosarcoma, grade 1. The tumor cells show both longitudinal and cross striations, pleomorphism and bizarre forms of nuclei. Inflammatory cells are seen scattered throughout the tumor. Hematoxylin and eosin stain; × 495.

From February 24 to March 18, 1943, the patient received high voltage roentgen therapy over the right antral region. With the use of 200 kilovolts, 20 milliamperes, 50 cm. distance, a half-value layer of 2 mm. copper, a filter of 2 mm. copper and 1 mm. aluminum and one anterior and one lateral portal each 3 cm. square, 200 r as measured in air was given to each portal twenty times in twenty treatment days, totaling 4,000 r to each portal. This gave a tumor dose varying from 4,000 to 6,000 r at the operative site.

The postoperative recovery during the following four weeks was unusually satisfactory. There was scarcely any elevation of temperature, and food intake was not a problem. The patient was discharged from the hospital at the end of five weeks and reported to the outpatient clinic thereafter for a total of thirty-two visits in 1943. During this nine month period, three small bony spicules were removed from the region of the hard palate remnants. At the end of six months the cavity could be easily visualized through the palatine defect and appeared clean.

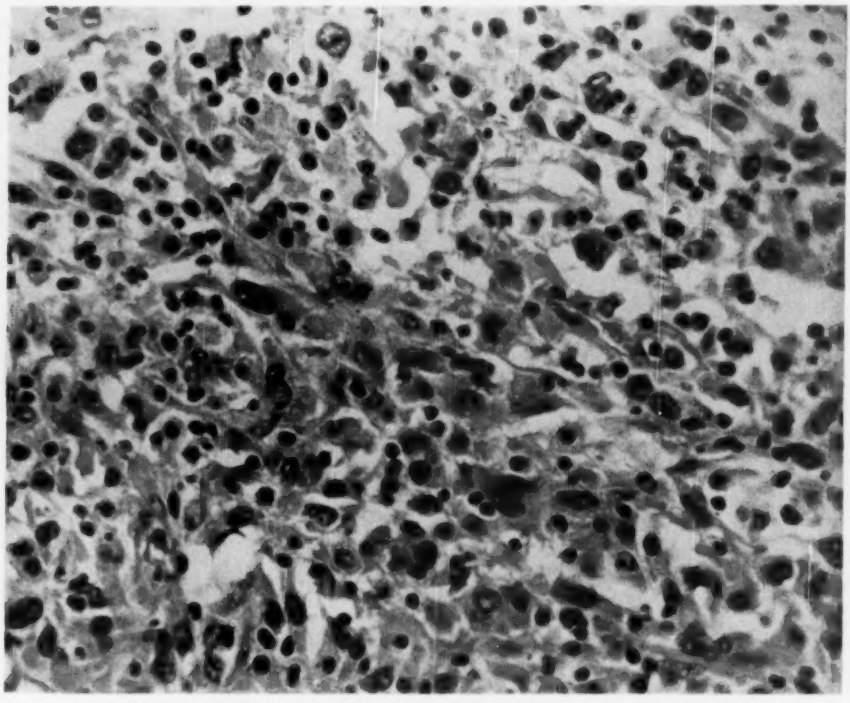

Fig. 2.—Rhabdomyosarcoma, grade 1, showing a mitotic figure, a multinucleated tumor giant cell with striations in the cytoplasm and a sheet of tumor cells of different shapes. Note the branching cytoplasm of the cells and the visible striations in most of the cells. Hematoxylin and eosin stain; × about 500.

There was a small (3 mm.) defect in the right nasal mucous membrane wall at the site of the natural opening of the nonexistent maxillary sinus. The patient has at all times been able to eat and drink without difficulty, the large palatine defect seemingly causing little or no discomfort and not requiring a prosthesis.

The patient resumed his employment in December 1943. He reported for reexamination in April 1944, November 1945, December 1946 and December 1949. The postoperative cavity has remained clean, and there has been no evidence of

recurrence of the original lesion. Roentgenograms of the skull, sinuses and chest in December 1949 showed normal structures except for the known defect in the right maxillary palatine region. Thus it would seem that at the time of this report, February 1950, the patient has remained free of demonstrable recurrence or metastasis of the original lesion for a period of seven years.

COMMENT

As previously noted, Stout's [5] classification of tumors derived from striated muscle is explicit and simple. He, along with Murphy, Dockerty and Broders [2]

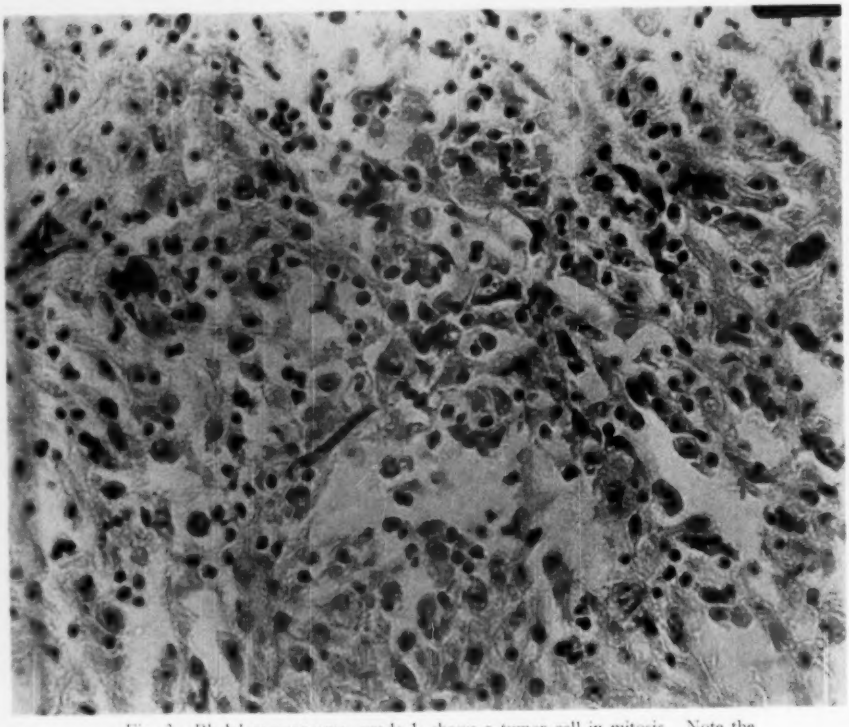

Fig. 3.—Rhabdomyosarcoma, grade 1, shows a tumor cell in mitosis. Note the striations in the cytoplasm of the tumor cells. Mallory's phosphotungstic acid and hematoxylin stain; × about 500.

and Khanolkar,[8] considers granular cell myoblastoma a more or less benign form and restricts the term rhabdomyosarcoma to the most malignant form. Jonsson [1] considered rhabdomyosarcoma the most malignant of soft tissue tumors and the most radioresistant.

8. Khanolkar, V. R.: Granular Cell Myoblastoma, Am. J. Path. **23**:721-739 (Sept.) 1947.

Striations in the cytoplasm of the tumor are not considered a prerequisite for the diagnosis as they may be absent. However, when a few cells show striations on special staining, the diagnosis is considered established. The tumor in the case presented here is interesting in that it shows striations (figs. 1, 2 and 3) and bizarre forms, tumor giant cells and mitotic figures (figs. 2 and 3). In brief it shows all the morphologic characteristics of rhabdomyosarcoma.

With a seven year cure, without signs, symptoms or indications of metastasis or recurrence and with the patient enjoying excellent health, we feel convinced that the line of treatment adopted in this instance, namely, extensive surgical intervention followed by irradiation, was the method of choice.

As far as we can determine, the origin of the tumor was in the right maxillary antrum, and no congenital muscular anomolies have been found by us in this region. The age of the patient (46) tends to rule against its being congenital in origin, and histologically we could not find the types of tumor cells described by Houette[9] and confirmed by Khoury and Speer,[10] that is, the blastoma cells of Masson, which are of small size, resembling lymphocytes. In the case under discussion, a large number of the cells showed striations and were uniformly large. Among these several areas showed cross striations as well as longitudinal striations. These findings and the clinical course encourage us to look on rhabdomyosarcoma as a disease entity with different grades of malignancy, grade 1 being the least malignant and grade 4 the most malignant. On the basis of this classification, we consider the tumor in the present case to be a grade 1 rhabdomyosarcoma.

SUMMARY

1. A case of rhabdomyosarcoma, grade 1, of the right maxillary antrum in a 46 year old Negro is reported. It is another instance of a patient with a neoplasm of the maxillary sinus whose presenting symptom, toothache, stimulated him to visit a dentist before seeking medical care. The history and the method of treatment, surgical extirpation followed by radium and high voltage roentgen therapy, are described. Photomicrographs showing both longitudinal and cross striations, bizarre nuclei and mitotic figures are included. The patient has been observed for seven years and seems in good health, without evidence of recurrence or detectable metastasis.

9. Houette, C.: Rhabdomyome diverticulaire congénital de la vessie, Ann. d'anat. path. **6**:267-282 (March) 1929.

10. Khoury, E. N., and Speer, F. D.: Rhabdomyosarcoma of the Urinary Bladder, J. Urol. **51**:505-516 (May) 1944.

Clinical Notes

HISTAMINE TREATMENT OF BELL'S PALSY

Report of Five Cases

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WINONA, MINN.

A case of Bell's palsy is seen by the average practitioner perhaps once every year or so. The patients are quite alarmed concerning the sudden onset of this condition and need reassurance plus some form of treatment, which, in the past, has usually been quite varied. Cecil [1] scarcely discussed treatment in the early editions of his book. Stevens,[2] in his twelfth edition, stated that little can be done, except in cases of syphilis, other than to remove the cause, if possible. He mentioned application of blisters near the stylomastoid foramen, the use of potassium iodide and electrical therapy. Hollender [3] added the use of strychnine, vitamin B, heat, massage and diathermy. Also recommended are strapping, splinting, wiring and, the most formidable of all, surgical decompression of the seventh nerve.

Medical literature shows only one article published on this subject. Skinner [4] very recently could find no publications on the subject, and I have found nothing since, although many are using histamine for this condition.

Skinner administered histamine in minute doses in three cases with quite phenomenal results. He expressed the belief that the cause is a vasospasm of the stylomastoid artery and its branches. Kettel [5] stated that Bell's palsy is due to a circulatory disturbance in which the nutrition of the nerve is affected, resulting in ischemic paralysis. The ischemia is the actual cause of the paralysis. Tickle [6] stated that although 85 to 90 per cent of patients with Bell's palsy recover, the remainder can be helped by the decompression operation if it is performed early. While results with histamine treatment are not completely certain, the advantages of a speedy recovery and the simplicity of treatment without operation seem undeniable.

In all five cases reported here there was peripheral involvement of the facial nerve.

Read at the monthly meeting of the Minnesota Academy of Ophthalmology and Otolaryngology, Dec. 9, 1949.

1. Cecil, R. L.: A Text-Book of Medicine, ed. 2, Philadelphia, W. B. Saunders Company, 1930.

2. Stevens, A. A.: A Manual of the Practice of Medicine, Prepared Especially for Students, ed. 12, Philadelphia, W. B. Saunders Company, 1928.

3. Hollender, A. R.: Office Treatment of the Nose, Throat, and Ear, Chicago, The Year Book Publishers, Inc., 1943.

4. Skinner, D. D.: Use of Histamine in Bell's Palsy, Ohio State M. J. **45**: 565-566 (June) 1949.

5. Kettel, K.: Bell's Palsy: Pathology and Surgery; Report Concerning Fifty Patients Operated on After Method of Ballance and Duel, Arch. Otolaryn. **46**:427-472 (Oct.) 1947.

6. Tickle, T. G.: Surgery of the Facial Nerve, Laryngoscope **55**:191-195 (May) 1945.

CASE 1.—M. L., a man aged 38, was first seen on Sept. 15, 1947 with a small mass easily discernible in the nasopharynx. The biopsy of this showed a pharyngeal bursitis (Thornwaldt's disease). Ten days later a sudden onset of facial paralysis developed on the left side. For five days he was given 100 mg. of nicotinic acid with no improvement. Then for three consecutive days he was given 250 cc. of histamine intravenously, after the method advocated by Horton; 1 mg. of histamine base is dissolved in 250 cc. of isotonic sodium chloride solution and given intravenously at a speed which just produces a comfortable flush without a headache. This may vary from 20 to 30 drops per minute up to as much as 80 or 100 drops per minute. The flow should be regulated to suit each individual patient. Improvement was noted within a few minutes after injection, beginning first in the eyelids.

The patient was able to close the eye completely but not so forcefully as the uninvolved eye. He lost a little ground between each treatment, but there was further recovery after the injection. The patient was using injections of nicotinic acid, 50 mm. daily, at home as a supplement. On October 18 he was completely recovered. In view of the results obtained in the later cases it is evident that more strenuous treatment should have been given here.

CASE 2.—H. W., a married woman aged 37, was first seen on Jan. 8, 1948, complaining that she had been unable to close her right eye for one day. She had the usual signs of facial paralysis. Treatment was begun the same day and continued for three consecutive days. Within a few minutes the patient could close her eye, and before leaving the corner of her mouth could be moved easily. Additional progress was made each day. Full recovery did not take place until January 28. There has been no recurrence.

CASE 3.—R. S., a married woman aged 49, experienced a sharp pain in the left ear on June 26, 1948. There was a little involvement of the facial nerve, which gradually became almost complete. Extreme pain, swelling and vesicles predominated early in the case, suggesting the diagnosis of herpes zoster oticus. She required several injections of morphine, sedatives for sleep and methadon by mouth, without any appreciable relief. Histamine treatment was begun on July 7 and continued for four days. Pain ceased quickly, and the paralysis began to improve, as in the preceding cases. Complete recovery was noticed on July 23. An interesting sidelight in this case was an occurrence of Bell's palsy on the opposite side in February 1944. The patient was treated with galvanic current, and after two months the condition cleared up with no residual paralysis.

CASE 4.—R. M., a man aged 42, complained, on July 20, 1948, of numbness in his left ear and inability to close the left eye for about a week. He also noticed swelling in back of the left ear for one day. He had the usual picture of Bell's palsy. Beginning on the first day he was given histamine intravenously in the same manner on four consecutive days. Improvement was rapid, and after four days treatment was discontinued. The patient gave himself injections of 1:10,000 dilution of histamine at home once daily for one week. He was apparently cured and was dismissed at this time.

CASE 5.—The last patient in the group, C. M., an unmarried woman aged 49, was seen Aug. 25, 1949 with a facial paralysis on the right of one week's duration. Prompt improvement was noted on the first intravenous injection of histamine. After four treatments she began using nicotinic acid, 50 mg. three times a day at home. A little residual paralysis was still present on September 13 and 19, and the patient was given 250 cc. of histamine on these dates. Complete recovery was noted on September 25.

SUMMARY AND CONCLUSION

Five patients with Bell's palsy are reported on, all treated by the daily office administration of 250 cc. of histamine intravenously in a dilution of 1:250,000

The response in all cases was prompt and complete. In evaluating treatment one must remember, however, that 85 per cent of such patients will recover spontaneously, although not completely. It is impossible to determine in advance just which patients are in the recovery group. Hilger[7] felt that the rate of spontaneous recovery is really closer to 50 per cent. It was gratifying to both the patient and the physician to be able to treat this condition with such simplicity and without complications of any kind. The use of histamine therefore seems warranted for patients who were formerly left untreated or who faced the prospect of surgical decompression.

51 East Third Street.

7. Hilger, J. A.: Personal communication to the author.

AN OPERATION FOR RELIEF OF STENOSIS OF THE STOMA FOLLUWING TOTAL LARYNGECTOMY

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NEW YORK

Stenosis of the tracheal stoma following total laryngectomy occurs when raw areas are left at the junction between the skin with the tracheal mucosa and when the trachea is insufficiently anchored to the skin, being allowed to retract downward. The stenosis is caused by the formation of constricting circular scar in the space between the tracheal rings and the skin surface. Occasionally, despite good surgical technic, stenosis ensues.

The operation described here has given satisfactory results. It is based on the following principles:

1. All of the scar tissue in the stenosed area (fig. 1, *a*) is excised.

2. The trachea is freed and raised: the tracheal stoma should be elevated to the level of the skin surface. This procedure, which was suggested by Dr. Joseph D. Kelly, is essential. In order better to anchor the trachea, it may be sutured to the adjacent strap muscles as well as to the skin.

3. The circular scar is eliminated by means of four stellate incisions radiating from the margin of the stoma and delimiting four flaps of skin, each of which is rotated (cart wheel fashion) around the defect (fig. 1, *b*, *c* and *d*). This procedure is similar to the Z flap used in plastic surgery for the relief of constricting scars around the extremities. It increases the amount of skin around the stoma, thus diminishing the chances for contraction (fig. 1, *d*).

4. Careful suture of the edges of the skin flaps to the edges of the tracheal mucosa is necessary to insure rapid healing and to minimize scar tissue formation.

After the operation a laryngectomy tube is worn by the patient until postoperative healing has occurred.

Results of this operation have been gratifying (fig. 2).

From the Plastic Surgery Clinic, Manhattan Eye, Ear and Throat Hospital, and the Plastic Surgery Unit, Department of Surgery, New York University-Bellevue Medical Center.

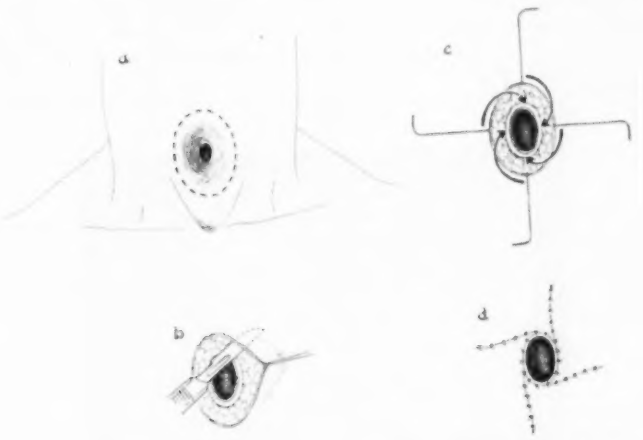

Fig. 1.—Technic of relief of stenosis by rotation flaps. (*a*) Outline of area of scar tissue to be excised. (*b*) The skin surrounding the defect is undermined. (*c*) Outline of the rotation flaps. (*d*) The flaps have been rotated around the defect. Careful suture of the skin flaps to the tracheal mucosa insures primary healing.

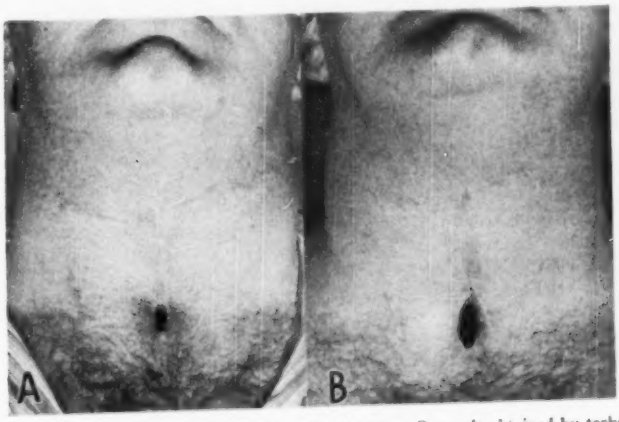

Fig. 2.—*A*, photograph of stenosed tracheal stoma. *B*, result obtained by technic illustrated in figure 1.

568 Park Avenue.

A SKIN RESISTANCE AUDIOMETER

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The greatest need in the field of pure tone audiometry is for a simple, reliable method of testing hearing by an objective response. By such a means one could evaluate hearing thresholds without having to depend on the patient's cooperation. Such a method would be of inestimable value in testing the hearing of infants and young children, particularly those for whom the failure to learn to speak has brought up the question of mental retardation.

All physicians are familiar with the frantic mother who brings her 2 year old child to the office to have his "hearing tested." He has not learned to formulate even the simplest words—even though he reacts to a loud handclap, the door buzzer, streetcar noises—and is now suspected of being feebleminded, with the prospect of several years' specialized management along the wrong lines.[1] Use of any device which would give an accurate estimate of the child's hearing for pure tones in the speech range would eliminate lost time and mental anguish. Such a procedure would also be useful in evaluating residual hearing with other conditions: congenital atresia of the auditory canals; hysterical deafness; psychogenic overlay of hearing impairment, and malingering.

A method of objective testing with a psychogalvanic response, utilizing the lie detector principle, has been described by Bordley and Hardy.[2] Although other methods have been described previously, this technic appears to be the first to give a reliable means of finding threshold levels.

There are five essential parts in the equipment used: (1) a standard pure tone audiometer; (2) an electric shock device; (3) a wheatstone bridge; (4) a DC amplifier, and (5) a recorder. This equipment operates on the principle that the resistance which the body offers to the passage of a minute direct electrical current varies with the activity of the sweat glands under the control of the sympathetic nervous system.

The patient is conditioned to a pure tone stimulus in the following manner: A tone of high intensity is delivered to the ear, followed in four or five seconds by a mild electric shock. This sequence is continued until the patient is conditioned; the conditioning is evidenced by a drop in skin resistance one and a half to two seconds after the sound, preceding the drop resulting from the shock. When the patient begins to respond consistently to this tone, the intensity of the sound is gradually diminished for each test sequence until threshold is reached, as shown by an absence in change of skin resistance, after delivery of the test tone.

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1. Hardy, W. G.: Relations Between Impaired Hearing and Pseudo-Feebleminedness, Nerv. Child. **7**:432-445 (Oct.) 1948.

2. Bordley, J. E., and Hardy, W. G.: A Study in Objective Audiometry with the Use of a Psychogalvanometric Response, Ann. Otol., Rhin. & Laryng. **58**:751 (Sept.) 1949.

The purpose of this paper is to show how the various pieces of equipment can be housed in a simple, small cabinet and operated electronically to make a simple, short procedure for one examiner.

Before the test is begun, the dials in the rear of the cabinet at the top are adjusted for length of signal tone and length of interval between signal tone and

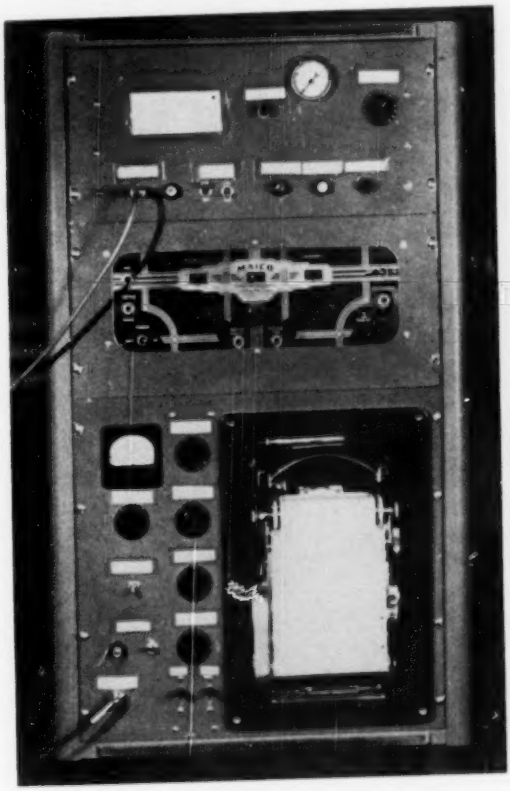

Fig. 1.—Front view of audiometer.

electrical stimulus, and the dials in the front of the meter at the top are adjusted for strength of the shock. These adjustments can be varied at any time during the test, and the strength of the shock can be measured on the meter without the shock reaching the patient.

The skin resistance electrodes are placed opposite each other on the palm and the dorsum or on the sole and the instep, the sites of greatest change in skin

resistance. The shock electrodes are placed on the arm, leg, or back. The bridge is balanced and the recorder started. The audiometer is set for the frequency and intensity of the sound desired, and the ear phone is held to the patient's ear by the nurse. Switch 1 is then closed; this procedure sets in motion the sound-interval-shock sequence, and the length of each is marked by a deflection in the

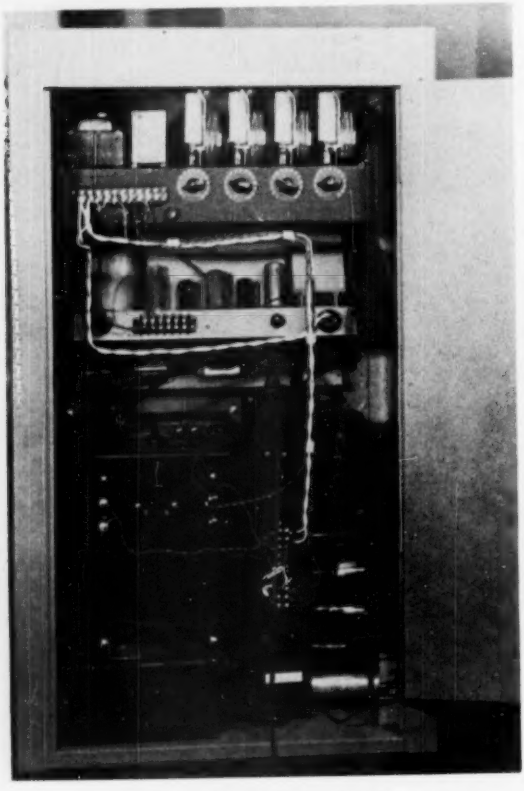

Fig. 2.—Rear view of audiometer.

ink line along the margin of the moving record. The sequence can be interrupted at any point by closing switch 2, which stops the shock in case the tone was not heard.

A voltage-regulating transformer has been built into the amplifier to reduce fluctuations due to line voltage changes that occur with nearby x-ray machines, etc.

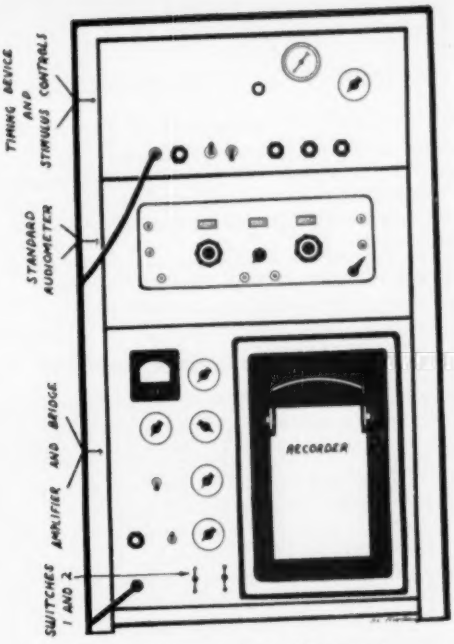

Fig. 3.—Diagram of front of instrument.

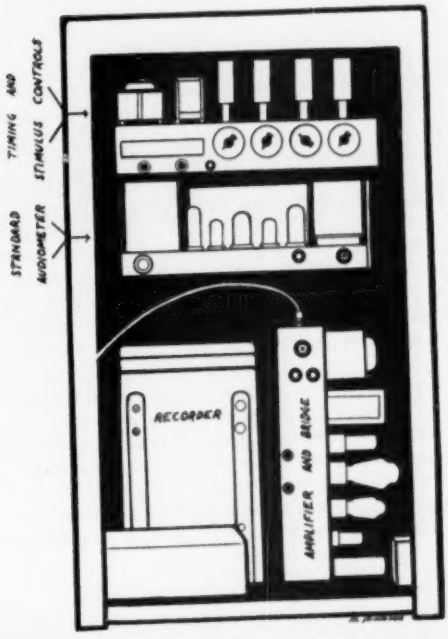

Fig. 4.—Diagram of rear of instrument.

SUMMARY

A modification of the equipment used in objective audiometry, as described by Bordley and Hardy, is presented. All variable factors are operated electronically so that each pure tone test sequence can be completed by the closing of a single switch. This procedure reduces the time required for the test and eliminates the need of an assistant.

Mr. Powel Crosley Jr. gave technical and financial assistance.

INFRACTION OF THE INFERIOR TURBINATES

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The practice of infraction toward the septum of the middle turbinate bone is widely known and is a method frequently resorted to in order to improve drainage of the paranasal sinuses of the anterior group, but one rarely hears of the process of deliberate displacement of the inferior concha.

It is often found that patients with septal deviation have enlarged turbinate bodies, in most cases the inferior on the concave side. This side is the larger and is rendered more or less normal by this "physiologic" hypertrophy. Otherwise, the natural respiratory functions of the nasal mucous membrane, i. e. warming, moistening and cleansing of the incoming air, would be continuously and adversely affected.

After correction of a septal deviation, the formerly narrow side is adequately distended, but frequently the originally large side has become too narrow. This condition is due to the above mentioned conchal hypertrophy. To save the patient and surgeon a second operation it is necessary to estimate the ultimate lumen on the concave side before beginning and to judge the space again immediately on completion of the septal correction. A small, inferior conchotomy on this side to compensate the septal displacement will produce the desired result. If the conchotomy is not performed at the same stage as the septal operation it is often necessary to do it later or to perform galvanocautery or submucous high frequency current coagulation on the hypertrophic inferior turbinate body.

It is for these reasons that the practice of infraction of the middle turbinate bone in sinus disease has been applied by me to the inferior concha to regain the desired space on the originally concave side. Thus, after the septal deformity is corrected, the inferior turbinate on the concave side is infracted toward the lateral nasal wall, after which procedure the nose is loosely packed with paraffin gauze on both sides or submitted to any other after-treatment selected for submucous septal resection.

This operation has substantially reduced secondary operations on the originally large side. It affords 1 to 4 mm. additional space at the narrowest spot and on the side referred to. The time required is, on the average, one minute, and only one additional and simple instrument, which will be described later, is used. After sufficient time has elapsed to prove the benefit of this operation, I have found that more patients have been wholly satisfied.

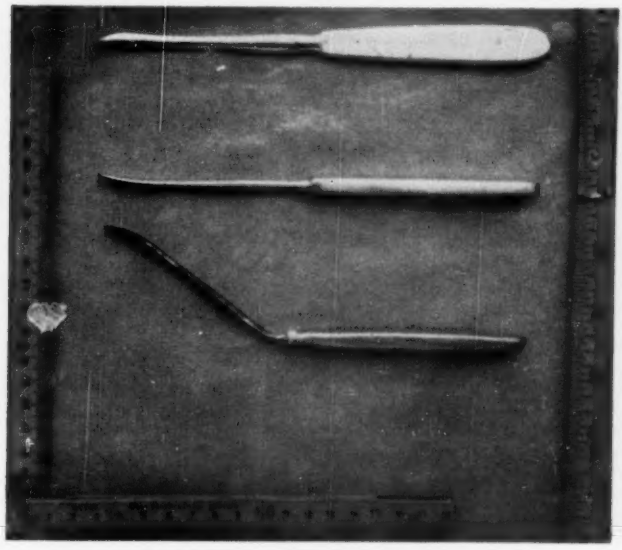
Gradually, this lateralization has also been applied to the originally convex side in many cases. Here, considerably less force is required, owing to the usually

atrophic condition of the inferior concha. The space gained is less: approximately 1 mm.

Since the bilateral infraction was introduced in June 1947, my assistants and I have performed it as a routine practice 158 times. It has proved entirely successful in every case but nine, in which it was found necessary to perform a secondary cautery of the inferior turbinate.

Subsequently, the operation was introduced in addition to cautery or coagulation of the inferior turbinate in most cases of hypertrophic rhinitis (turbinal hypertrophy or hyperplasia). In this connection, it was practiced 166 times on 89 patients.

Observations made during the actual practice of this operation have led to the conclusion that when adhesions between the inferior turbinate and the septum are



Frontal and lateral view of two standard Langenbeck elevators (top two) and lateral view of an angular Langenbeck elevator (bottom).

divided, final infraction of the former hinders their reuniting and thus facilitates and shortens after-treatment.

TECHNIC

When the infraction is performed in addition to or as a substitute for galvanocautery the anesthesia as commonly used for the latter procedure is satisfactory, and this usage also applies to the obliteration of synechiae. When it is performed as the final phase of a submucous septal resection, and especially when the major operation was of long duration, it is desirable to inject 2 to 3 ml. of a 1 per cent procaine hydrochloride solution with epinephrine along the line of attachment when the septal correction is almost completed.

Infraction of the inferior turbinate bone requires more force and a stronger instrument than is necessary for the middle concha, owing to the stronger structure of the inferior turbinate and, in a much lesser degree, probably to its more curved line of insertion. A Langenbeck elevator (dissector) is used, but any slender, tapering, blunt-pointed instrument with a slight curve at the end will serve the same purpose. In the figure a photograph of three dissectors is shown. The top two are standard Langenbeck elevators, the angles of view differing 90 degrees. However, it is probable that the same instrument with the handle bent at about 45 degrees would be found more convenient because the visibility of the working end would be improved (third instrument in the figure). I have not yet sufficiently tested the probable advantages of this suggested alteration.

The method of procedure is now as follows: The instrument is held like a pencil, with the point directed toward the turbinate to be infracted, that is, with the convexity to the septum. The thumb is on the conchal side. The right hand is used for the right concha and inversely. The point is placed on the most prominent part of the concha and then pressed laterally under visual control. If the turbinate does not yield with a slight cracking sound the elevator is taken out and grasped in the full hand like a tennis racket, again with the thumb on the conchal side; the instrument is then reintroduced and pressed again. In more than half of all the cases the bone will succumb to the first method. An occasional turbinate bone may require considerable force with the second grip. However, most turbinate bones will be infracted rather easily, either with the first or the second method. I prefer always to try the former method first. After having made a dent in the most prominent part (usually one third or halfway within the nose) repeat the same procedure behind and in front of the first dent, slight cracks and visible displacement of the turbinate bone being the result. It is preferable to use an instrument with a slightly curved end and to let the point work against the concha. If the convex side of the instrument is used the force required for infraction is, in my opinion, often too great. The same is true of a flat instrument. In both cases a larger part of the turbinate bone must be infracted in one movement. Working with the blunt point and establishing dent after dent facilitates the operation considerably.

As a rule, the operation is bloodless, but occasionally a few drops of blood will flow. In itself it requires no after-treatment; any treatment considered necessary after a concurrent operation will be easily tolerated.

COMMENT

Advantages of the Described Procedure.—The operation is practically always bloodless and leaves little or no wound. The mucous membrane is left intact to perform its respiratory function to the full extent. It takes little time and only one additional and simple instrument. In fact, it is so simple and harmless that it has been gradually introduced as a routine procedure bilaterally after septal resection and during galvanocautery and after division of synechiae on the side under treatment.

Disadvantages of the Described Procedure.—Theoretically, there is a slight danger of fracturing the lateral nasal wall (laterad to the free part of the inferior turbinate). This occurrence could cause hemorrhage, for instance, in the antrum, or damage to the lacrimal duct or sphenopalatine vessels and nerve. However, so far none of these mishaps has ever occurred in my department. The force

required to fracture the lateral nasal wall appears to be much greater than that necessary to infract the free blade of the inferior turbinate, and this is to be expected for anatomic reasons. Moreover, the surgeon is warned that he must cease to continue the pressure on a cracking sound and with the feeling that the free blade is collapsing, much as in the case of puncture of the antrum through the inferior meatus. Third, the operator can see the conchal displacement.

The space between the inferior turbinate and the lateral nasal wall will be lessened, and this may sometimes cause a little difficulty in case antral lavage is required. Therefore I do not perform the operation when there are signs of antral disease. If this situation is suspected the operation may be postponed until the sinus is cured, or, if strictly necessary, another distending procedure is adopted. If there is little room between the turbinate and the lateral nasal wall, either because of natural causes or previous infraction (and this proves an obstacle to puncture of the antrum) it is quite a simple matter to (re)infract the head of the turbinate toward the septum.

ORIGIN

As mentioned before, I have borrowed the idea from infraction of the middle turbinate. Having no free access to literature, I cannot trace when and by whom infraction of the middle concha in sinus disease was introduced. It is, perhaps, for the same reason that I have never found any treatise on infraction of the inferior turbinate. Certainly, it is not mentioned in the current textbooks. My friend and colleague, Dr. Bruining, of the Batavia Military Hospital, devised the same principle independently of me. He began the operation with a cut of about 1 cm. in the proximal end of the line of the turbinate's attachment to facilitate the infraction. The cut was made with a Struycken conchotone, but I have never found this cut necessary. Later on, the cut was dropped and infraction achieved with the closed long blades of a Killian nasal speculum. This instrument has the disadvantage of not having a curved end and therefore, in my opinion, requires more force. Both Dr. Bruining and I were already performing this operation before the last war.

SUMMARY

Infraction of the inferior turbinate toward the lateral nasal wall is described as a simple procedure to relieve blockage of the nasal passages. As a rule, it is practiced additionally to another distending operation (septal correction, galvanocautery), but it may suffice alone in some cases. It is also of value as a final phase in obliteration of nasal synechiae.

The turbinal mucous membrane remains completely undamaged. The disadvantages are rare, and they are only slight difficulties in antral puncture.

The only additional instrument needed is a Langenbeck or similar dissector.

NOTE—Since submission of this paper for publication, Evans has published a report on the same principle.[1] The reader is referred also to a paper by Tremble.[2]

Djalan Dago 102.

1. Evans, J. M.: Role of Compensatory Hypertrophy and Simple Atrophy in Intranasal Surgery, Arch. Otolaryng. **50**:172 (Aug.) 1949.

2. Tremble, G. E.: Mechanics of the Nose, Arch. Otolaryng. **51**:205 (Feb.) 1950.

TOOTH GUARD FOR USE WITH DAVIS MOUTH GAG

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AND
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It is an almost universal practice to use a Davis mouth gag to obtain exposure in performing tonsillectomies and adenoidectomies with the patients under general anesthesia. The Davis mouth gag has many advantages, the foremost of which are excellent exposure, control of tongue and preservation of adequate airway. The greatest disadvantage is the possibility that the gag may loosen or knock out a newly erupted maxillary incisor tooth or a deciduous tooth. Sooner or later one who performs tonsillectomies is faced with the embarrassing situation of explaining

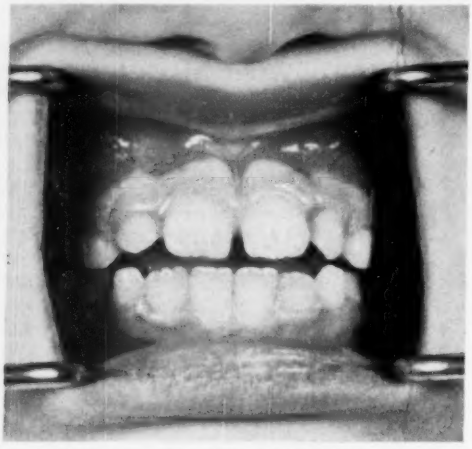

Fig. 1.—Newly erupted, poorly supported, central incisors.

tic loss of an incisor tooth to the parents or the patient. Anterior deciduous teeth are easily detached. Premature loss of one of them may complicate the occlusion and cause a drifting of teeth. Newly erupted permanent teeth have practically no root structure, and they are extremely easy to knock out. The root of a permanent incisor is not completely formed until about four years after the initial eruption. If a maxillary central incisor is lost prematurely, it is practically impossible to construct a satisfactory space maintainer which could be worn until the adjoining teeth are sufficiently mature to support a fixed bridge. Unless a fixed bridge or space maintainer is constructed, the adjoining teeth will drift into the space and a major orthodontic problem will result. Prosthetic replacement is very unsatisfactory in young patients.

A search of the literature failed to reveal reports of an adequate method of protecting vulnerable maxillary incisors. A rubber padding may be used on the tooth plate of the Davis gag to protect the enamel of the teeth, but accidental dislodgment of a maxillary incisor tooth can still occur.

The purpose of this presentation is to report on the use of a tooth guard which will protect the maxillary incisors during tonsillectomies and adenoidectomies. About eight years ago the need for such a device was seen at this institution. The eye, ear, nose and throat and dental departments designed a satisfactory splint, or

Fig. 2.—Tooth guard of hard dental baseplate, showing application of carborundum on heated biting surface and as adapted to cast of upper jaw.

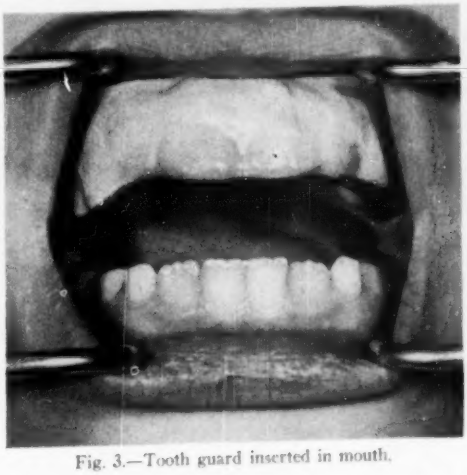

Fig. 3.—Tooth guard inserted in mouth.

tooth guard,[1] which has been in constant use since that time and has proved very satisfactory.

1. Dr. Leslie Smith, now chief of the Eye, Ear, Nose and Throat Department, United States Marine Hospital, New Orleans, La., cooperated in this study.

A definite preoperative routine has been developed at this institution for tonsillectomies and adenoidectomies. In addition to the general medical survey, blood studies and chest roentgenogram, it includes an order to the dental service to construct a tooth guard. The actual construction of the tooth guard is a relatively simple technical matter. A dentist should be consulted who understands the problem at hand. It is a simple matter for him to construct such a tooth guard. A beeswax impression of the maxillary teeth from the second bicuspids or first molars is obtained. This impression is poured in dental stone or plaster. Shellac wax baseplate is adapted on a wet stone model. After the tooth guard is trimmed neatly, as shown in figure 2, a suture or dental floss may be attached to the prosthesis for ease of handling. It is unnecessary to process the baseplate in a permanent material such as acrylic. However, this may be done, particularly if the tooth guard is to be used for protection of the teeth during laryngoscopy or bronchoscopy. The shellac wax baseplate is sufficiently strong for use with a Davis mouth gag, and it will insert into the mouth with ease. Recently carborundum powder has been dusted on the heated occlusal and incisal surface of the prosthesis. The carborundum adheres firmly to the heated surface, and the excess is washed off. This provides a nonskid surface on which the Davis mouth gag grips. This feature alone is worth the trouble of construction of the tooth guard. The cost of construction of the prosthesis or tooth guard should be minimal, and the cost cannot be compared with the dangers of accidental loss of an anterior tooth during operation.

The use of the above described tooth guard in no way complicates the use of the Davis mouth gag but facilitates its use and does not reduce the amount of exposure in even the smallest mouths. Occasionally, when only one central incisor is erupted, a mouth gag is difficult to anchor, and the tooth guard provides a firm anchoring surface.

It is not claimed that the tooth guard is a panacea that allows for complete forgetfulness of the incisor teeth, nor is it a substitute for careful handling and manipulation at the time of surgery, but its use is advocated for protection of the teeth during a tonsillectomy and adenoidectomy.

A NEW NASAL SPLINT

IRVING B. GOLDMAN, M.D.
NEW YORK

The nasal splint described here differs from other nasal splints of this type in the following manner.

1. There is a left and right thread; thus, when the center screw is turned, pressure is applied equally to both nasal bones.

2. The screw is turned from the top, preventing a shift in position or movement of the splint while pressure is being applied.

3. The center overhead band produces a perfect fit and prevents the splint from slipping on the nose.

From the Division of Rhinoplasty of the Mount Sinai Hospital.

Presented at the meeting of the American Otorhinologic Society for the Advancement of Plastic and Reconstructive Surgery, Inc., in Atlantic City, June 6, 1949.

4. The lateral bands are supplied with hooks which fit into the side bars. These bands can be removed and washed.

The precautions observed in the use of clamps should be followed, especially when used immediately postoperatively. The osteotomy should be complete for application of this splint to a greenstick fracture would be disastrous. The skin

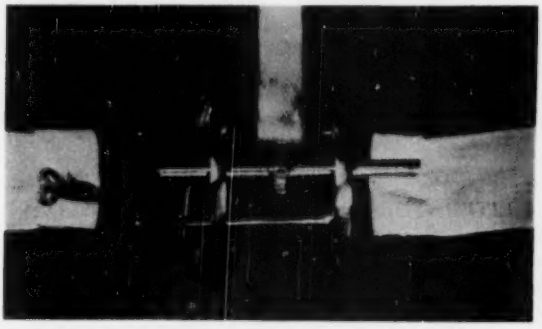

Fig. 1.—Photograph of splint showing right and left threads, top screw and position of the bands.

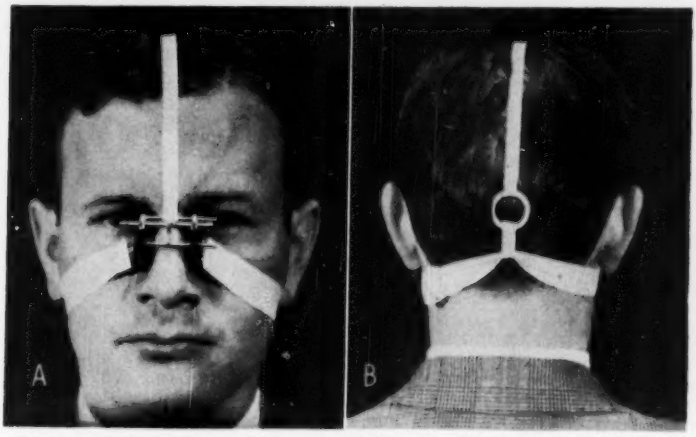

Fig. 2.—*A*, anterior view of splint in position. *B*, posterior view. Note snugness of the bands, which prevent splint from slipping.

of the dorsum must be elevated in the proper plane. Obviously, if the separation is too near the surface, pressure may cause necrosis of the skin.

In the hands of men who use a clamp routinely, this type of prefabricated splint has its place.

121 East Sixtieth Street.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology for 1947 and 1948

PLASTIC SURGERY

LYNDON A. PEER, M.D.
JOHN C. WALKER, M.D.
AND
JOHN VAN DUYN, M.D.
NEWARK, N. J.

THE LITERATURE on plastic surgery for 1947 and 1948 contains many interesting and valuable articles which should be read in the original. Otolaryngologists may build up a good yearly reference file by writing to selected authors for reprints of their papers.

EDITORIAL COMMENT ON CURRENT TRENDS IN THE LITERATURE

Otolaryngologists have been opposed to nasal operations in infants and young children because of the belief that such procedures would interfere with normal growth of the nose.

In an editorial comment in the 1946 review, we stated that simple fracture and realinement of the bony bridge or conservative adjustment of a displaced septal cartilage did not, in our experience, stunt or adversely affect normal growth of the nose.

We also presented case evidence that one might safely repair bifid nose in young children by suturing the lateral and alar cartilages together to obliterate the median cleft, by fracture and narrowing of the broad bony bridge and by insertion of a segment of the child's rib cartilage to fill the saddle depression.

It will be noted that all these operations are conservative procedures. They effect a change in the position of the bones and cartilages of the nose from the abnormal to the normal without actual removal of any cartilage or bone, or the nose is added to when rib cartilage is inserted to fill a saddle depression.

We advised against removal of the septal cartilage or of the alar and lateral cartilages except in the case of a much displaced lower end of the septal cartilage, in which it might be necessary to remove a small segment of cartilage in order to provide adequate breathing space. This removal should be conservative since very little is actually known about the so-called growth centers in the nasal cartilages.

Articles appearing in the 1947 and 1948 literature advise that complete removal of the septal cartilage and radical nasoplasty in grow-

ing children will not interfere with normal growth of the nose. This dangerous doctrine is presented without any long term observation and case reports.

The members of our department advise otolaryngologists against extensive or complete removal of the nasal septum or removal of the lateral and alar cartilages in growing children.

Complete removal of the nasal septum is not advisable at any age.

AUTOGENOUS CARTILAGE AND BONE GRAFTS

Cartilage and bone have a rather simple structure in that both are composed of their respective parenchymal cells, which are embedded in and surrounded by a large amount of nonliving intercellular substance. This inanimate intercellular substance, which is the product of activity of the living cells, determines the character and physical properties of these tissues.

Thus, cartilage is firm but relatively elastic owing to a gelatinous intercellular substance, and bone is rigid, owing to a calcified material between its cells.

The survival of the intercellular substance in cartilage and bone grafts after transplantation depends on the survival of the living elements in the grafts, the cartilage cells and the bone cells. If the cells survive as living entities, the intercellular substance will remain; if the cells die, the intercellular substance will eventually be invaded and absorbed.

Cartilage.—When the cells in a cartilage graft fail to survive transplantation (which seldom happens), or when they have been killed by heat before transplantation, the dead gelatinous matrix remains for long periods. Dr. Gordon New made practical use of this physiological fact when he advocated the use of "boiled" autogenous cartilage grafts, which have less tendency to bend or become distorted after transplantation. There is, however, a slow but progressive invasion and replacement of these dead autogenous cartilage grafts by host fibrous tissue, which are often associated with new bone formation. Living autogenous cartilage grafts are, therefore, the better grafting material.

Bone.—Mowlem demonstrated that the cells in cancellous bone grafts survive in the nasal tissues even though the graft is not in contact with bone, whereas the cells in dense cortical bone grafts not in contact with bone die and the calcified matrix of the graft is slowly absorbed.

One of us (L. A. P.) demonstrated that the cells in nasal septal bone grafts not in contact with bone survive transplantation and that the grafts retain their intercellular structure as septal bone.

It seems evident, therefore, that septal bone grafts are good grafting material for small defects, and the use of cancellous bone grafts is advisable in the repair of large defects.

The question arises concerning the fate of dense cortical bone grafts which are in contact with the nasal bones at one end and with soft tissues at the other end. The cells in these grafts usually fail to survive transplantation because of nutritional difficulties due to the dense structure of the bone. The graft is believed to be replaced, however, by infiltration of host cells from the nasal bones or from their periosteum (creeping substitution).

One may summarize the facts concerning living autogenous cartilage and bone grafts as follows:

Since both living autogenous cartilage grafts and living autogenous septal and cancellous bone grafts survive transplantation, all may be used for grafting purposes. Most American plastic surgeons prefer cartilage, while in England plastic surgeons appear to favor the use of bone.

When we state that living autogenous cartilage grafts survive transplantation as living tissues, we refer to septal, alar and auricular cartilage as well as to rib cartilage.

The presence or absence of perichondrium does not appear to affect the survival of cartilage grafts.

SURGERY OF THE EYELID

Any surgeon doing careful reconstructive or plastic procedures on the eyelid is indebted to Wheeler and Hughes for their contributions to this type of work. This observation was emphasized by Sherman [1] when he discussed his experiences in the army.

Dupertuis [2] is of the opinion that, when possible, the utilization of lid tissue for eyelid reconstruction provides the best result and that in the split lid technic the insertion of a free clavicular skin graft into the new lid improves its appearance and position. For more serious deformities involving bony loss, pedicle grafts are indicated; these grafts are planned to minimize any scar deformity on adjacent donor sites. Split mucous membrane from the lips can be obtained simply and in adequate quantity to provide the preferred substitute lining for the anophthalmic socket. A thoughtful plan of reparative surgery to include all damaged areas adjacent to, or associated with, eyelid deformities will eliminate duplication of operative procedures and speed ultimate successful reconstruction.

A proper conception of these principles was demonstrated by Callahan [3] in an excellently illustrated article. He had corrected multiple lid deformities, in a case, with a free skin graft, a pedicle flap, intermarginal adhesions and wiring of the canthi.

1. Sherman, A. E.: Reconstruction of the Eyelid, Arch. Ophth. **39**:427 (March) 1948.

2. Dupertuis, S. M.: Eyelid Reconstruction in the Blinded, Plast. & Reconstruct. Surg. **3**:269 (May) 1948.

3. Callahan, A.: Severe Lid Deformities, Plast. & Reconstruct. Surg. **3**:456 (July) 1948.

Spaeth [4] discusses the repair of upper eyelids in patients in whom more tissue has been lost than can be restored by excision of scars and local undermining. The patients are divided into two main groups, those in whom the eye is normal and those in whom it is absent. In the former group, missing conjunctiva must be replaced by conjunctiva swung up from the lower lid, by a free graft or by oral mucous membrane. On the anophthalmic patients, split grafts may be used instead of mucous membrane.

Spaeth describes a method to repair complete loss of the upper lid, an operation in which part of the conjunctiva and tarsus of the lower lid and all the skin are used. A direct suture of the two lids is done after the edges of the remnants of the upper lid have been freshened and the rim of the lower lid completely undermined. Later, the palpebral fissure is restored, thereby leaving the skin and most of the tarsus, with its attached conjunctiva, on the new upper lid; the lost skin of the lower lid is restored by a free skin graft.

A case in which severe ectropion of the lower lid is treated by a pedicle graft from the upper lid is presented by Stallard,[5] of England.

To correct a crescentic deformity of the lid margin, Fox [6] takes a graft from the upper portion of the eyebrow to fill in the defect.

A new application of an old technic was made by Armstrong and Garcia [7] for the reconstruction of total or partial loss of the lower lid. A free composite graft was taken from the crus of the helix and carefully sutured in the defect.

Kiskadden and McGregor [8] describe a procedure for correcting coloboma of the eyelid and, in an illustrative case, show a patient with an excellent result. Descriptions of four other methods for correction are included for comparison.

Two cases of melanosis of the eyelids are reported by Braithwaite.[9] The author concludes that acquired melanosis of the eyelid is precan-

4. Spaeth, E. B.: The Reconstruction of the Upper Lid, Surg., Gynec. & Obst. **84**:804 (April 15) 1947.

5. Stallard, H. B.: Ectropion Corrected by Bridge-Pedicle Graft, Brit. J. Plast. Surg. **1**:77 (July) 1948.

6. Fox, S. A.: Crescentic Deformities of the Lid Margin, Arch. Ophth. **39**:542 (April) 1948.

7. Armstrong, C. J., and Garcia, F. A.: The Use of a Composite Graft in Reconstructive Surgery of the Lower Eyelid, Plast. & Reconstruct. Surg. **3**:79 (Jan.) 1948.

8. Kiskadden, W. S., and McGregor, M. W.: Coloboma of the Eyelids, Plast. & Reconstruct. Surg. **2**:60 (Jan.) 1947.

9. Braithwaite, F.: Melanosis of the Eyelids, Brit. J. Plast. Surg. **1**:206 (Oct.) 1948.

cerous and at its appearance the lesion should be treated by surgery or radiation or both. Cure is possible. He states that congenital blue nevi may become locally invasive, so they should be excised in childhood.

In a case of bilateral ectropion of the four eyelids due to ichthyosis congenita, successful repair (by isografts of the same blood groups) was carried out by Spaeth and Cappriotti.[10]

(Comment.—It is known that isogenous skin grafts [skin transplanted from one patient to another] may "take" primarily, but they tend gradually to disappear and be replaced by scar tissue or by the patient's own skin in about two months. This change occurs regardless of the blood grouping, and the only exception is in the case of identical twins. The successful isogenous skin grafting, reported by Spaeth and Cappriotti, therefore is a rare exception to the usual occurrence.)

SURGERY OF THE EAR

In 1943, Peer [11] reported a new method for reconstructing an auricle by inserting "diced cartilage grafts" in a perforated vitallium ear mold and burying the mold with its cartilage filler beneath the patient's abdominal skin.

In a recent article,[12] he describes his later experience with this method of repair, which has been used in 22 cases. Description of the technic follows.

First Operation.—Rib cartilage is removed from the right side of the patient's chest and diced into many fine cubes. These diced cartilage grafts are introduced into each half of a perforated vitallium ear mold, and the two halves of the mold are fastened together, thereby pressing the diced cartilage grafts into the shape of an ear. The mold with its cartilage filler is inserted in a pocket beneath the patient's abdominal skin (fig. 1). During a period of five months, blood vessels and connective tissue grow through the openings in the mold and fasten the diced cartilage segments firmly together.

Second Operation.—The vitallium ear mold is removed from the abdominal pocket five months after burial, the two halves of the mold separated and the diced cartilage ear framework removed from the mold. This diced cartilage ear framework is inserted in a pocket beneath the skin in the ear region (fig. 2) and a firm dressing applied to keep the skin in contact with the cartilage.

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10. Spaeth, E. B., and Cappriotti, O. A.: Heteroplastic and Isoplastic Skin Grafts, Plast. & Reconstruct. Surg. **3**:707 (Nov.) 1948.

11. Peer, L. A.: Diced Cartilage Grafts, Arch. Otolaryng. **38**:156 (Aug.) 1943.

12. Peer, L. A.: Reconstruction of the Auricle with Diced Cartilage Grafts in a Vitallium Ear Mold, Plast. & Reconstruct. Surg. **3**:653 (Nov.) 1948.

Third Operation.—The diced cartilage framework is dissected free from the temporal fascia and the periosteum of the temporal bone. A split skin graft sutured over a dental stent mold is inserted behind the new auricle to cover the back surface of the diced cartilage framework and the raw surface of the scalp. This procedure will permit the new auricle to stand out at an angle from the side of the head.

Fourth Operation.—The distorted ear lobe is shifted into normal relationship with the reconstructed auricle.

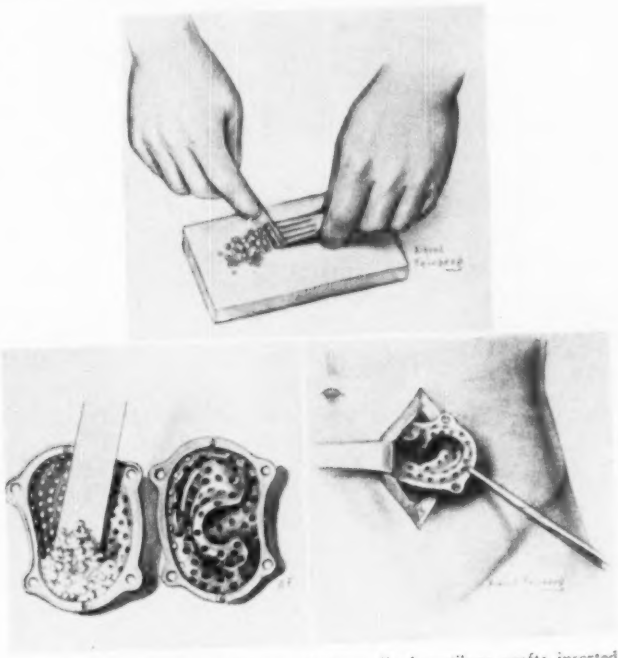

Fig. 1.—Formation of ear framework from diced cartilage grafts inserted in vitallium ear mold. (From Peer.[12])

Fifth Operation.—Final trimming is done to sharpen the helix, increase the depth of the concha and canal region and remove the hair-bearing skin.

Peer states that whenever possible the first operative step to reconstruct an auricle should begin when a child is about 4 years of age. This allows the surgeon a sufficient interval of time in which to complete the ear before the child enters school, thus lessening his handicap and facilitating adjustment with schoolmates.

Aufricht [13] modified Peer's method of total ear reconstruction in several ways, the modification based on his interpretation of the auricle not as a shell but as a semicircular body. He uses an acrylic mold with small holes, filling the mold with flat chips and thin platelets of cartilage rather than "diced" pieces. The acrylic mold does not include a helix, which is later supplied by a skin tube, nor is the posterior surface lined

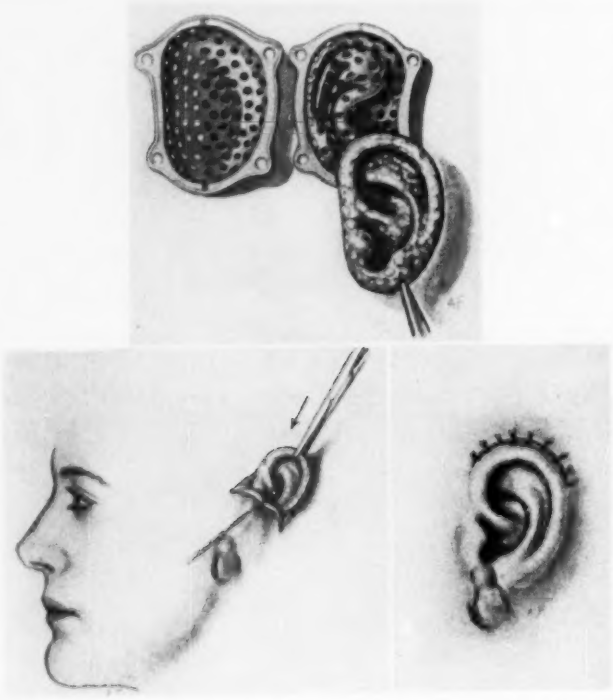

Fig. 2.—Removal of diced cartilage ear from mold and transplantation in ear region. (From Peer.[12])

with a split graft left attached to the head. The technic is given in detail, along with photographs of one patient.

During the past two years, Conway and his colleagues [14] have had experience with 10 cases of partial and complete reconstruction of the

13. Aufricht, G.: Total Ear Reconstruction: Preliminary Report, Plast. & Reconstruct. Surg. **2**:297 (July) 1947.

14. Conway, H.; Neumann, C. G.; Gelb, J.; Leveridge, L. L., and Joseph, J. M.: Reconstruction of the External Ear, Ann. Surg. **128**:226 (Aug.) 1948.

external ear. Technical details in the cutting and carving of costal cartilage are described. The authors advance the opinion that if attention is given to these details, reconstruction of the external ear can be effected that will be acceptable to patient and surgeons.

A case of unilateral congenital absence of the auricle is reported by Ford.[15] He reconstructed the auricle out of materials taken from the sound ear, plus a skin-tubed flap taken locally. The materials from the sound ear included skin and cartilage from the posterior region and a wedge-shaped piece of ear substance from the middle portion.

Rubin and his co-workers[16] present one case in which they used polyethylene for reconstruction of the ear.

(Comment.—The search for a "perfect foreign body" which will remain permanently in the soft tissues is as old as medical history; thus far, however, no foreign substance has been discovered which is as satisfactory as a graft of the patient's own living tissue. Polyethylene, like vitallium and tantalum, appears to be an inert substance, but proof is lacking that it will be tolerated in human tissues for long periods of time.)

Steffanoff[17] and Lewin[18] advocate utilizing local tissue (auriculomastoid tubes) for reconstruction of partial loss of the auricle or helix.

A method used successfully in four cases of cicatricial atresia of the ear canal and one case of congenital webbing is described by Steffensen.[19] The lateral skin surface of the membrane is incised to create quadrants; internal and external flaps are made; and, finally, the flaps interdigitated so as to cover all raw surface—an application of the Z-plastic technic.

RHINOPLASTY

In a very readable article, Safian[20] sounds a note of warning to young plastic surgeons about the confusing statements and unfounded conclusions in some of the recent literature on rhinoplastic procedures. He quotes a number of passages to illustrate his point and comments in

15. Ford, J. F.: A Method of Plastic Reconstruction of the Auricle: Preliminary Report, Plast. & Reconstruct. Surg. **1**:332 (Nov.) 1946.

16. Rubin, L. R.; Robertson, G. W., and Shapiro, R. N.: Polyethelene in Reconstructive Surgery, Plast. & Reconstruct. Surg. **3**:586 (Sept.) 1948.

17. Steffanoft, D. W.: Auriculo-Mastoid Tube Pedicle for Otoplasty, Plast. & Reconstruct. Surg. **3**:352 (May) 1948.

18. Lewin, M. L.: Reconstruction of the Helix, Arch. Otolaryng. **47**:802 (June) 1948.

19. Steffensen, W. H.: A Method of Correcting Atresia of the Ear Canal, Plast. & Reconstruct. Surg. **1**:329 (Nov.) 1946.

20. Safian, J.: A Critical Review of Recent Literature on Rhinoplasty, Plast. & Reconstruct. Surg. **2**:463 (Sept.) 1947.

a humorous as well as a serious vein. He concludes with the following advice, which certainly applies to more than just the field of plastic surgery:

> I wish to urge every serious-minded young man who intends to specialize in this [rhinoplastic] branch of plastic surgery to learn to distinguish between that which is essential to the performance of his duty to his patient, and that which is done too frequently either to "glamourize" the surgeon or confuse the operator.

(Comment.—This is an important article which should be read by all otolaryngologists interested in rhinoplastic surgery. Safian criticizes a growing trend in publications on nasal plastic surgery in which the writers make simple surgical problems so difficult to understand that even the experienced plastic surgeon cannot comprehend their meaning. The editorial boards of our surgical journals should not accept these articles for publication.)

Gatewood [21] outlines, with drawings (fig. 3), a method of rhinoplasty in which a chisel is used instead of a saw, both for removing the hump and for narrowing the bony sides of the nose. He believes that the use of the chisel rather than the saw saves time and energy, leaves the bone surfaces smooth and clean, traumatizes the soft tissues less and leaves no bone dust to produce osteogenesis.

(Comment.—Surgeons experienced in nasoplasty are about evenly divided in their preference for the use of the saw and the chisel. We generally use the chisel. Good results are obtained by both methods.)

Ersner and Alexander [22] call attention to their method for inserting a columellar strut. They insert the strut in an almost completely closed bed between the columellar cartilages. They like to use live cartilage taken from the patient's septum at the time of operation. In no instance has absorption, disintegration or extrusion of the strut occurred.

It is Lefkon's belief that rhinoplastic procedure and external removal of the septal cartilage in children may be safely undertaken when they are at an early age without in any way impairing the future development of the organ.[23] According to Lefkon, it is a fact that transplanted autogenous or preserved cartilage remains alive and is not absorbed.

(Comment.—This article advises extensive or complete removal of the septal cartilage in young children without including any long term case reports to demonstrate that such a procedure does not impair the future development of the nose. The author, therefore, makes a statement

21. Gatewood, W. L.: Substitution of the Chisel for the Saw in Reconstructive Surgery of the Nose, Plast. & Reconstruct. Surg. **2**:149 (March) 1947.

22. Ersner, M. S., and Alexander, M. H.: A New Technique for the Insertion of a Columellar Strut, Eye, Ear, Nose & Throat Monthly **26**:257 (May) 1947.

23. Lefkon, I. M.: Rhinoplasty in Children, Arch. Otolaryng. **48**:73 (July) 1948.

of fact which is not supported by proof. The members of our department advise against complete removal of the septal cartilage in growing children—or in adults. [See editorial comment.] Lefkon states that "transplanted autogenous or preserved cartilage remains alive." How can preserved cartilage in which the cells are dead at the time of transplantation remain alive after transplantation?)

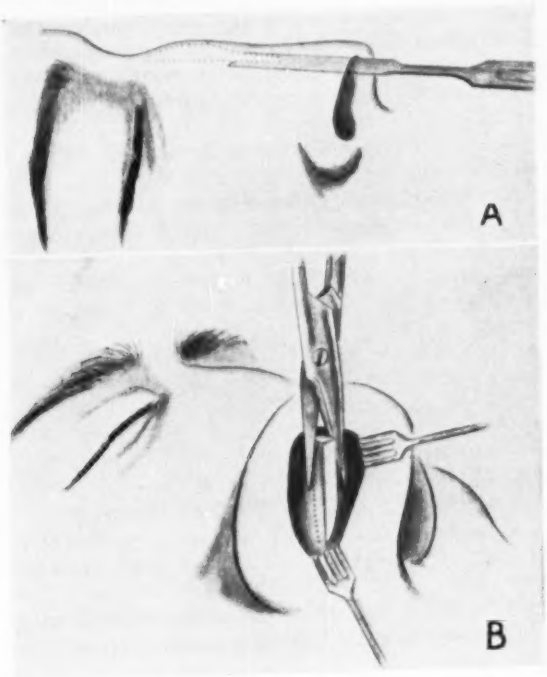

Fig. 3.—*A*, removal of dorsal elevation with chisel. *B*, excision of anterior part of septum to shorten nose. (From Gatewood.[23])

Fomon's [24] summary of his article on plastic repair of the obstructing nasal septum follows:

Generally speaking, a better result will be obtained if the rhinoplasty and the septal correction are done in separate operative stages three or more weeks apart. Inasmuch as the final outcome of the operation depends in a measure on the healing qualities of the tissues, the smaller amount of trauma inflicted by dividing the stages speaks for less reparative reaction and a better anatomic and physiologic result.

24. Fomon, S.; Gilbert, J. G.; Silver, A. G., and Syracuse, V. R.: Plastic Repair of the Obstructing Nasal Septum, Arch. Otolaryng. **47**:7 (Jan.) 1948.

As to the order of sequence, the old teaching advocated that the rhinoplasty precede the submucous resection on the grounds that the septum supported the nose and that if the submucous resection were done first a subsequent removal of a hump might open into the resected area and result in saddling. New concepts of the structural anatomy of the septum have proved this assumption fallacious, and today the submucous resection is done first since it permits an easier osteotomy and a more symmetric alinement of the pyramid.

(*Comment.*—Fomon states that new concepts of the structural anatomy of the septum have demonstrated that it [the septal cartilage] is not an important supporting structure for the dorsum of the nose. These so-called "new concepts" have all been postulated by Fomon and his co-workers and have not been accepted by the majority of experienced otolaryngologists and plastic surgeons.

The members of our department advise against complete removal of the septal cartilage in rhinoplastic surgery.)

The anatomic and surgical relationship between septal and external nasal deformities are discussed and septal deformities classified by Maliniac.[25] He advises certain principles of repair for these different deformities. In the timing of repair, if a deflection interferes with successful performance of rhinoplasty, it should, as a rule, be corrected simultaneously with, or prior to, the rhinoplasty.

In an absence of the columella resulting from syphilis, Shaw and Fell [26] used a tube graft made between the thumb and index finger. They stated that a very good match in color was obtained by this procedure.

For total loss of the columella, Salinger [27] moves an acromiopectoral tube to the base of the columella by way of the angle of the jaw and the nasolabial fold. The results in three cases are illustrated.

The successful use of cancellous bone grafts from the ilium in reconstructing the depressed nasal bridge in five cases is reported by Suggit.[28] Postoperative roentgenograms confirmed the good position of the grafts.

Holt and Lloyd [29] suggest the use of methyl methacrylate as an implant in rhinoplastic work. This is the material commonly known as lucite.[8]

(*Comment.*—Why not use autogenous cartilage or bone grafts instead of a foreign body?)

25. Maliniac, J. W.: Role of the Septum in Rhinoplasty, Arch. Otolaryng. **48**:189 (Aug.) 1948.

26. Shaw, M. H., and Fell, S. R.: Columella Reconstruction, Brit. J. Plast. Surg. **1**:111 (July) 1948.

27. Salinger, S.: Reconstruction of the Columella, Plast. & Reconstruct. Surg. **2**:87 (Jan.) 1947.

28. Suggit, S.: The Repair of the Depressed Nasal Bridge, J. Laryng. & Otol. **60**:501 (Dec.) 1946.

29. Holt, J. A. B., and Lloyd, R. S.: Rhinoplasty: The Use of Methyl Methacrylate Implants, Arch. Otolaryng. **47**:406 (April) 1948.

For severely comminuted depressed fracture of the nose with involvement of the lateral processes and septum, Maliniac [30] uses a combination of endonasal and externasal splinting. After preliminary reduction, the crushed dorsum is held up by perforated acrylic splints inserted high along the dorsum on each side. To these acrylic splints are attached wires threaded on straight needles, which, after being passed through the dorsum, are attached to a notched rod on the external splint. The external splint is a bilateral compressor (see figure [30]).

The chief advantages of this method are simplicity and effectiveness, the caved-in septum being elevated by the same procedure. The nasal passages remain free during treatment, no packing being required.

SURGERY OF THE FACE AND SCALP

The literature on the correction of facial paralysis is reviewed by Owens.[31] He concludes that the best results are obtained by the utilization of muscle combined with support. This method gives the greatest degree of reanimation to the face. According to Owens' technic, the muscle used is the masseter and the support, fascia lata. Three fascia lata strips are attached at one end to the anterior half of the masseter muscle, and at the other end, to the upper lip, the angle of the mouth and the lower lip respectively. Eleven cases are reported in this article, and three more are added in a subsequent one.[32]

For facial nerve paralysis, Brown and MacDowell [33] advise two autogenous fascia lata strips running from the temporal muscle to the middle of the upper lip and the angle of the mouth respectively. In some cases additional fascial slings are required for the cheek, the lower eyelid or, occasionally, for the eyebrow.

Schuessler's technic (described in 1944) for the repair of facial paralysis with tantalum wire is discussed by Schultz and Fowler.[34] The authors found a tendency of the saddles to cut through the muscle bundles. To overcome this tendency they advocate the use of tantalum

30. Maliniac, J. W.: Compressive Suspension Splint for Severely Comminuted Fractures, Plast. & Reconstruct. Surg. **2**:331 (July) 1947.

31. Owens, N.: Implantation of Facial Strips Through the Masseter Muscle for Surgical Correction of Facial Paralysis, Plast. & Reconstruct. Surg. **2**:25 (Jan.) 1947.

32. Owens, N.: Revaluation of the Implantation of Fascial Strips Through the Masseter Muscle for Surgical Correction of Facial Paralysis, Ann. Rhinol. & Laryng. **57**:55 (March) 1948.

33. Brown, J. B., and MacDowell, F.: Support of the Paralyzed Face by Fascia, J. A. M. A. **135**:18 (Sept. 6) 1947.

34. Schultz, L. W., and Fowler, E. F.: Treatment of Facial Paralysis by Tantalum Wire Suspension, Plast. & Reconstruct. Surg. **2**:538 (Nov.) 1947.

gauze screening instead of the saddles. This gauze may be suspended by the tantalum wire. Schultz and Fowler point out the simplicity of this procedure but warn against the material being placed too near the skin. Only a few case reports are included, and these are relatively new.

In the correction of facial defects, local flap shifts and modifications of the Z-plasty are considered the methods of choice by Macomber and Berkeley.[35]

The use of neck tube pedicle skin is indicated for almost any defect of the face, forehead or oral cavity requiring skin and subcutaneous tissue from a distant point, provided that the defect is not too large for the amount of neck tissue available.

The advantages of using tubed pedicle skin from the neck are as follows: (1) better color and texture match; (2) production of minimal defect in donor area; (3) ideal thickness; (4) proximity of tissue to be moved, and (5) great vascularity.

Mills and Patterson[36] report a series of cases with photographs illustrating the use of Z-plastic and sliding flaps and the principle of multiple excision in dealing with scar and nevus deformities about the face and neck. They urge that ample intervals of time be allowed between steps, for this is the greatest ally in the softening and relaxation of tissues.

In the opinion of Brown and Fryer,[37] it is impossible to separate repairs of soft tissues and underlying bony framework. The authors carry out repair in the first 24 hours, if possible. If the patient is seen after this, it is probaly best to wait until subsidence of the swelling. Rarely should repair be delayed longer than from seven to 10 days.

They consider the use of nerve blocks best for anesthetizing except for the endotracheal method for children and advise against depending on roentgenograms for diagnosis.

Débridement of both soft parts and bone is advocated. This should be done very sparingly. They use 000 white silk for buried sutures, and 000 black silk for skin suturing and emphasize the importance of using bulky pressure dressings.

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35. Macomber, W. B., and Berkeley, W. T.: Use of Neck Tubed Pedicles in Reconstruction of Defects of the Face, Plast. & Reconstruct. Surg. **2**:585 (Nov.) 1947.

36. Mills, J. T., and Patterson, J. B.: The Combined Use of Multiple Excision, the Z-Plastic and Sliding Flaps in the Correction of Certain Congenital and Acquired Anomalies, Plast. & Reconstruct. Surg. **2**:249 (May) 1947.

37. Brown, J. B., and Fryer, M. P.: Management of Compound Facial Injuries, Am. J. Surg. **76**:625 (Nov.) 1948.

A case of extensive damage to tissues of the forehead and bridge of the nose is reported by Adams.[38] After temporary repair by the use of a split skin graft, the area eventually was entirely covered by shiftings of the normal forehead skin. The depressed bridge of the nose was filled out with a pickled cartilage graft.

The interesting problem of scalp losses is discussed at length by Kazanjian and Webster [39] in a well illustrated article.

The undertaking of immediate treatment depends on whether or not the periosteum is intact. If so, immediate skin grafting is done, a fairly thick split graft from elsewhere on the body being used. The authors state, however, that the free scalp is utilizable provided all subcutaneous fascia and fat are removed with care. Otherwise, only the hair is of value.

Where the cranium is denuded of periosteum, the outer cortex should be chiseled away until multiple bleeding points appear, and after a week or slightly longer the area is ready for a split graft. This method is more rapid than that of drilling multiple burr holes.

If the whole scalp cannot be grafted at once, the anterior part is chosen first because of the effect of contractures.

The main complications of early inadequate therapy are, Kazanjian and Webster continue, contracture, scar breakdown and carcinomatous degeneration. Scar breakdown is common following scalp losses due to the very slow return of sensation to this area. The treatment of these complications may require large tube pedicle grafts migrated from a distance.

For repairing a defect caused by complete loss of the cheek, Hadjistamoff [40] uses the skin over the remainder of the jaw plus neighboring neck skin, which includes a trapezium-shaped flap attached to it and extending downward. This trapezium-shaped flap is turned up under the neck and under the jaw skin above it and sutured in place so as to serve as a lining. The outer part of the wound is then closed transversely by simply undermining the double pedicle of neck and jaw skin from chin to ear. Later, the base of the trapezium-shaped lining is severed and turned back to cover a remaining bare area.

In a well illustrated article, New [41] again emphasizes the value of his sickle flap in the repair of defects of the face. For reconstructing the tip of the nose and in total rhinoplasty, he now uses a midline rather than a lateral attachment of the flap in order to prevent a one-sided pull.

38. Adams, W. M.: The Use of Neighboring Tissues in the Correction of an Extensive Facial Deformity, Plast. & Reconstruct. Surg. **2**:105 (March) 1947.

39. Kazanjian, V. H., and Webster, R. C.: The Treatment of Extensive Losses of the Scalp, Plast. & Reconstruct. Surg. **1**:360 (Nov.) 1946.

40. Hadjistamoff, B.: Restoration of the Cheek by Using the Skin of the Jaw-Neck Region, Plast. & Reconstruct. Surg. **2**:127 (March) 1947.

41. New, G. B.: Further Uses of the Sickle Flap in Plastic Surgery, Plast. & Reconstruct. Surg. **1**:271 (Nov.) 1946.

Chronic fissure of the lower lip is considered inconsequential and benign, but it is very annoying to the patient who has one. Dingman [42] excises the lesion and closes it on the modified Z principle.

Réthi [43] (of Budapest) describes a simple way of closing retroauricular fistula following radical surgery on the ear. He swings a small circumscribed flap over the opening and then pushes back the auricle to cover this flap.

Berson [44] describes a method of removing rhinophyma without the necessity of applying a split graft or allowing the raw surface to granulate. He excises beneath the skin and utilizes this to cover the defect as if it were a flap.

By means of facial prints, Rubin [45] came to the conclusion that Langer was correct in that a scar parallel to the lines would be inconspicuous because it resembled a line but that he was incorrect when he said that the lines run in the same direction as the muscles. Actually, the skin lines are at right angles to muscle pull.

A case of congenital defect of the scalp is reported by Peer and Van Duyn [46] which terminated fatally owing to detachment of the crust and hemorrhage from the superior longitudinal sinus. It is suggested that in such cases an attempt be made to cover the area of the defect by scalp flaps before necrosis of the membrane takes place.

Depression of the floor of the orbit and displacement of the lower orbital margin should be corrected as soon as the initial reaction to the trauma has subsided. In late cases of depressed floor, however, it is not possible to elevate the floor to its normal position because of firm adhesions. Sherman [47] believes that cancellous iliac bone is preferable to all other substances as material for an implant. Tantalum and acrylic resin, being preformed, may not give the proper contour. Cartilage, whether autogenous or preserved, does not become attached to the bone and may change after operation.

A diagnosis of depressed fracture of the floor of the orbit, in Sherman's opinion, is based on a history of trauma and the presence of depression of the eyeball, with some enophthalmos.

42. Dingman, R. O.: Chronic Fissure of the Lower Lip, Plast. & Reconstruct. Surg. **3**:613 (Sept.) 1948.

43. Réthi, A.: Operative Treatment of the Retroauricular Opening Remaining After Radical Operation of the Ear, Plast. & Reconstruct. Surg. **3**:371 (May) 1948.

44. Berson, M. L.: Rhinophyma, Plast. & Reconstruct. Surg. **3**:740 (Nov.) 1948.

45. Rubin, L. R.: Langer's Lines and Facial Scars, Plast. & Reconstruct. Surg. **3**:147 (March) 1948.

46. Peer, L. A., and Van Duyn, J.: Congenital Defect of the Scalp, Plast. & Reconstruct. Surg. **3**:722 (Nov.) 1948.

47. Sherman, A. E.: Reconstruction of Orbital Floor Defects, Surg., Gynec. & Obst. **84**:799 (April 15) 1947.

After an experience with 117 iliac bone grafts in filling in facial defects, Macomber [48] expresses his "unqualified satisfaction with cancellous iliac bone." Tantalum, acrylic resin, fascia lata, fat, deepithelized skin or cartilage is not as uniformly acceptable. Cancellous bone is probably as rapidly vascularized as a free skin graft and is actively octogenic, becoming firmly attached to surrounding structures and developing a cortex under the connective tissue investment. It is most useful in defects and depressions of the jaw, orbital rim, forehead and nose.

SURGERY ON THE MANDIBLE AND THE MAXILLA

Blocker [49] believes that the increasing popularity of cancellous bone as the material of choice for relief from large and small bony defects of the mandible is fully justified. Cancellous bone very quickly develops strength and evidence of firm bony union within eight to 14 weeks after operation, when dentures may usually be fitted. For extensive defects such as angle-to-angle loss of bone, the author uses a tantalum tray containing several block grafts, which are packed around with bone chips.

Macomber and his associates [50] present data on 92 patients on whom mandibular bone grafts were carried out. They feel that grafts taken from the tibia, ilium or rib can adequately restore bony continuity of the mandible. A good occlusional splint used to maintain mandibular fragments is described. The authors stress the importance of soft tissue work preoperatively. The important factors are (1) complete absence of residual infection; (2) adequate splinting; (3) adequate preoperative and postoperative care, and (4) painstaking surgical technic.

In a study of 34 cases of jaw deformities, in all of which large bone graft replacements were required, Soderberg and Mulvey [51] reach certain conclusions. After mobilization of fragments (by traction or, as a last resort, surgical procedures), stabilization was usually maintained by intraoral splinting combined with an acrylic resin or silver saddle. Occasionally, combined intraoral and extraoral splinting was used. Bone grafts were cancellous bone from the iliac crest, either completely or partially decorticated and with or without additional cancellous bone chips beneath the transplant. The distances covered varied from 1 cm.

48. Macomber, D. W.: Reconstruction of Bony Defects of the Face, Surg., Gynec. & Obst. **83**:761 (Dec.) 1946.

49. Blocker, T. G.: Cancellous Bone Grafts to the Jaw, Surg., Gynec. & Obst. **84**:553 (April 15) 1947.

50. Macomber, W. B.; Shepard, R. A., and Crofut, V. E.: Mandibular Bone Grafts, Plast. & Reconstruct. Surg. **3**:570 (Sept.) 1948.

51. Soderberg, B. N., and Mulvey, J. M.: Mandibular Reconstruction in Jaw Deformities, Plast. & Reconstruct. Surg. **2**:191 (May) 1947.

to half the mandible. The rule was to wait for a recovery period of six months before beginning work. The period for postoperative splint fixation was eight weeks for small transplants and at least 12 weeks for the larger ones.

Successful use of an iliac bone graft (autogenous) to restore continuity in a case of nonunion of the mandible following fracture is reported by Parker.[52] He believes that the use of iliac grafts is the method of choice in such cases.

When removal of a section of mandible is necessary, or when traumatic loss has occurred in children or adentulous adults, splinting or interdental fixation may be unsatisfactory. In such cases Byars [53] drills holes in the ends of the bone fragments and inserts a stainless steel bar to bridge the defect. This procedure permits secondary repair of the defect by a bone graft without the necessity of a preliminary laborious restoration of the fragments to their original relationship. The bar is left in place until it is certain that restoration of the fragments to their proper positions can be easily done at the time of the bone grafting.

Vitallium plates have been used by Freeman [54] for immobilization of the bone ends after resection of the mandible. This procedure is advocated as a simple, dependable method of stabilization in aged or debilitated persons in whom bone grafts are not feasible and as a temporary measure for those patients who will eventually have bone grafts.

A case in which the nose, lip and maxilla were restored by operation and prosthesis is reported by Kazanjian.[55] He believes that surgical restoration should be undertaken whenever possible. Acrylic material or vulcanite rubber is, in his opinion, preferable for artificial noses because they are solid enough to permit efficient supporting attachments.

Campbell [56] reports an interesting case in which he reconstructed the left maxilla, using a temporal muscle flap to fill out the depression, as described by Gillies in 1919. Campbell used a palate flap to close the palatal defect and iliac bone to fill out the depression further.

52. Parker, D. B.: Fracture of the Mandible with Non-Union and Bone Graft, J. Oral Surg. **5**:171 (April) 1947.

53. Byars, L. T., and McDowell, F.: Preservation of Jaw Function Following Surgery and Trauma, Surg., Gynec. & Obst. **84**:870 (April 15) 1947.

54. Freeman, B. S.: The Use of Vitallium Plates to Maintain Function Following Resection of the Mandible, Plast. & Reconstruct. Surg. **3**:73 (Jan.) 1948.

55. Kazanjian, V. H.: Restoration of Nose, Lip and Maxilla by Surgery and Prosthesis, Plast. & Reconstruct. Surg. **2**:531 (Nov.) 1947.

56. Campbell, H. H.: Reconstruction of the Left Maxilla, Plast. & Reconstruct. Surg. **3**:66 (Jan.) 1948.

Dingman [57] believes that the surgical correction of prognathic defects of the mandible has a greater possibility of success if the defects are operated on through the body of the mandible rather than the ramus. A two-stage procedure is presented by the author. It preserves the inferior alveolar canal contents.

Marino and Craviotto [58] treated micrognathia in a young girl successfully by the use of extraoral fixation, ladder section of bone with the immediate filling in of space with cancellous bone chips and the closing off communication to the oral cavity, leaving a small drain to the outside. By the extraoral fixation, postoperative vomiting is not obstructed; later, intraoral wiring is done for better immobilization. Immobilization was maintained for 40 days. The result was a functional as well as a cosmetically acceptable jaw.

An article on ankylosis of the temporomandibular joint by Straith and Lewis [59] is very complete, including the history, etiology and pathology of the disease. The authors state that conservative treatment is useless. They create a false joint and make a "hockey stick" incision in front of the ear for exposure. They emphasize the importance of dental care and after-correction of the chin.

In a series of 27 patients with limited mouth opening, Brown and his associates [60] found eight with actual bony ankylosis involving the coronoid process. Surgical correction consisted of removal of the coronoid process and enough of the surrounding displaced bone to prevent reunion. The procedure is the same for both osseous and extensive fibrous ankylosis. The intraoral approach is preferred because it avoids the facial nerve and the parotid gland structures. Postoperative jaw exercises are important; the simple use of a rubber block and active motion usually suffice.

HARELIP AND CLEFT PALATE

Peer [61] prefers to repair harelip with light ether anesthesia when the child is six weeks old. He closes palatal clefts usually when the patient is 18 to 20 months of age. Stress is placed on proper speech training and orthodontic care.

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57. Dingman, R. O.: Surgical Correction of Developmental Deformities of the Mandible, Plast. & Reconstruct. Surg. **3**:124 (March) 1948.

58. Marino, H., and Craviotto, M.: Micrognathia: Treatment with External Prosthesis, Plast. & Reconstruct. Surg. **2**:260 (May) 1947.

59. Straith, C. L., and Lewis, J. R.: Jr.: Ankylosis of the Temporo-Mandibular Joint, Plast. & Reconstruct. Surg. **3**:464 (July) 1948.

60. Brown, J. B.; Peterson, L.; Cannon, B., and Lischer, C.: Ankylosis of the Coronoid Process of the Mandible (and Associated Scar Limitation of Jaw Function), Plast. & Reconstruct. Surg. **1**:277 (Nov.) 1946.

61. Peer, L. A.: Management of Cleft Palate Deformities, J. New Jersey State Dental Soc. **19**:21 (Nov.) 1947.

In repair of hairlip, Callister,[62] of the University of Utah Medical School, describes a stay suture that extends through the septum on the side of the intact nostril, through a soft rubber catheter and on through the alar cartilage and skin on the affected side and back to prevent lateral creeping of the alar cartilage.

Kazanjian [63] finds that as patients with cleft lips grow up, deformities develop which were not prominent when the original correction was made. The commonest of these later deformities are unevenness of the vermilion border, flatness and tenseness of the upper lip, nasal tip distortion and deviation of the nasal septum. Another serious secondary deformity of harelip and cleft palate is due to lack of growth of the maxilla. This is more marked in patients who have been operated on in childhood.

Kazanjian believes that when the upper lip is underdeveloped the lower lip becomes overdeveloped to compensate. In all cases in which there is retraction and underdevelopment of the upper lip, he transfers a section of the lower lip to the upper (Estlander-Abbe operation, fig. 4). The details of the operation are given and the results fully illustrated. A dental appliance to support the upper lip is later made for these patients.

In repairing cleft palates by the Wardill procedure, Havens [64] emphasizes the importance of extending the dissection beyond the base of the hamular process. He also brings out the advantages of using the Veau operation in anterior clefts of the palate and of the alveolar process. This operation may be combined with correction of single or double cleft lips.

In another of their beautifully illustrated articles, Dorrance and Bransfield [65] clearly explain their conception of the action of the superior constrictor muscle in effecting velopharyngeal closure. The external fibers, which bring the posterior wall of the pharynx forward, are attached anteriorly to the hamular processes; the internal fibers, which serve as the actual sphincters, are inserted in the soft palate. Fracture and backward displacement of the hamular process, as advised in push-back operations, serve two purposes: One is to return the external

62. Callister, A. C.: A Technique Designed to Prevent Lateral Creeping of the Alar Cartilage in the Repair of Harelip, Plast. & Reconstruct. Surg. **3**:617 (Sept.) 1948.

63. Kazanjian, V. H.: Estlander-Abbe Operation in Treating Secondary Harelip Deformities and Defects of the Upper Lip Resulting from Cancer, Plast. & Reconstruct. Surg. **2**:307 (July) 1947.

64. Havens, F. Z.: Cleft Lip and Palate, Arch. Otolaryng. **48**:9 (July) 1948.

65. Dorrance, G. M., and Bransfield, J. W.: Studies in the Anatomy and Repair of Cleft Palate, Surg., Gynec. & Obst. **84**:878 (April 15) 1947.

fibers of the superior constrictor muscle to this more posterior and hence more nearly normal position; the other is to destroy the power of the tensor palatine muscle by turning it into a levator muscle. The postoperative hole in the center of the soft palate is less often seen when this procedure is carried out adequately.

Baxter and Cardoso [66] believe that following their modification of the push-back operation for short palates, speech results are definitely better than those following the ordinary operative procedures. In the first stage, when the palate flap is delayed, the raw surfaces are covered

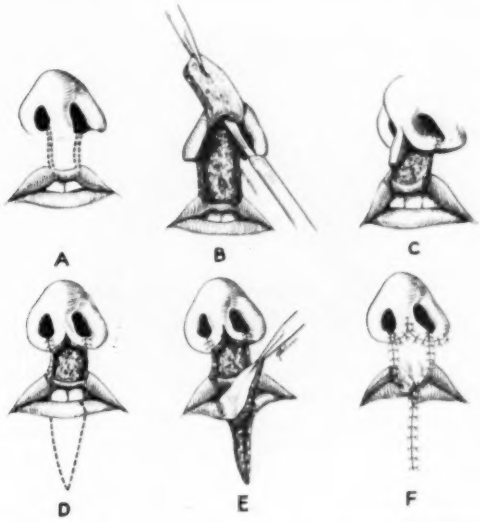

Fig. 4.—Diagrams, *A, B, C, D, E* and *F*, showing the various stages of operation for the elevation of the tip of the nose to improve the contour of the nostrils and for the transfer of a triangular flap from the lower lip to the upper lip. (From Kazanjian.[63])

with a split graft applied by means of a buried dental compound stent. A complete take is the usual result. In the second stage, two to three weeks later, the push-back procedure is done, a splint being used for pressure only if the lateral margins of the flap cannot be conveniently approximated. The authors believe that postoperative massage is beneficial, starting four to six weeks after the second stage.

66. Baxter, H., and Cardoso, M.: A Method of Minimizing Contracture Following Cleft Palate Operations, Plast. & Reconstruct. Surg. **2**:214 (May) 1947.

Ludwick [67] (of Lincoln, Nebraska) gives a clear presentation of Nebraska's plan of incorporating dental care and rehabilitation of speech in the program by the State Services for Crippled Children. He stresses the importance of not dropping such cases when operation is completed.

LARYNX AND PHARYNX

Following the pioneer work of King, in 1939, Clerf [68] now uses mobilization and lateral fixation of an arytenoid muscle as an excellent procedure for improving the airway in bilateral stenosis of the larynx. Only the unilateral operation should de done, since such adduction as occurs during phonation is seen only on the side not operated on.

Fig. 5.—Pharyngeal bursa and cleft palate. (From Adair.[71])

Vaughan [69] discusses the surgical correction of nasopharyngeal atresia, considering both the congenital and the acquired types. Where scar tissue is present, satisfactory flaps of palatal mucous membrane cannot be made, and in this situation the excellent Makenty operation is not feasible. If the scar is not heavy, the adhesions between the soft palate and the nasopharynx are freed and a silver diaphragm interposed for

67. Ludwick, P. G.: The Rehabilitation of Cleft Palate Patients, Am. J. Orthodontics **34**:235 (March) 1948.

68. Clerf, L. H.: The Surgical Treatment of Paralytic Stenosis of the Larynx, Plast. & Reconstruct. Surg. **2**:326 (July) 1947.

69. Vaughan, H. S.: Nasopharyngeal Atresia, with Report on New Procedure by Skin Graft to Lateral Pharyngeal Wall, Plast. & Reconstruct. Surg. **1**:309 (Nov.) 1946.

four weeks. Where there is so much scarring present that extensive raw areas result from the separation, skin grafting is used in addition.

A method of treating cicatricial stenosis of the nasopharynx, the result of an experience with 37 cases, is outlined by Figi.[70] In seven of the cases the stenosis was complete. With local anesthesia, he incises the stricture transversely and inserts a split graft tied about a sponge rubber mold in which tubes for breathing have been placed. After the grafts have taken, a self-retaining obturator of acrylic resin is used to minimize secondary contracture.

Adair [71] reports two cases of pharyngeal bursa associated with cleft palate (fig. 5). This lesion is a midline diverticulum from the posterior wall of the nasopharynx, extending from the lower border of the pharyngeal tonsil to the periosteum of the occipital bone; it is not a true bursa.

Many bursae are asymptomatic and are never diagnosed. Clinical symptoms of infection of the bursa include a characteristic midline occipital headache, postnasal discharge, which may be purulent or mucoid, and low grade fever. The most characteristic symptom is periodic dropping down of hard cone-shaped crusts from the nose into the throat.

Any treatment is unsatisfactory. Heavy curettement with adenoid curets is best.

SKIN GRAFTING

Greely [72] presents an article of 10 pages on skin grafting. No new contributions are included, but the article is excellent for a general report on the subject or as a reference for surgeons who do not use skin grafts routinely.

As viewed by New and Devine,[73] the dissected full thickness graft taken from behind the ear or from the upper eyelid is the finest graft available for any small, surgically clean facial wound. The use of a thick split graft is indicated in the release of contractures; thin split grafts are used as linings and as coverings for large granulating surfaces; hair-bearing scalp flaps for restoring a normal hair line; the sickle forehead flap for the reconstruction of half a nose or a tip, and the cervical flap for some defects of the lower half of the face in men. The use of the skin near the sternoclavicular junction for certain defects on the face and neck is discussed.

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70. Figi, F. A.: Cicatricial Stenosis of the Nasopharynx: Correction by Means of a Skin Graft, Plast. & Reconstruct. Surg. **2**:97 (March) 1947.

71. Adair, M.: Pharyngeal Bursa Associated with Cleft Palate: Report of Two Cases, Plast. & Reconstruct. Surg. **3**:478 (July) 1948.

72. Greely, P. W.: Skin Grafts, S. Clin. North America **28**:113 (Feb.) 1948.

73. New, G. B., and Devine, K. D.: Skin Grafting Methods and Their Indications, S. Clin. North America **26**:890 (Aug.) 1946.

In Brown and Cannon's opinion[74] composite free grafts find their greatest use in losses about the tip of the nose, the ala and the columella. The contained cartilage supplies an internal stretch of the skin and support while the blood supply is being established around the edges. The graft must fit the defect accurately, and a firm pressure dressing is necessary. The height or distance from the nutrient edge is a factor.

Fogh-Anderson,[75] of Denmark, gives an interesting history of the Estlander-Abbe operation and points out that the Danish surgeon Stein was probably the first to perform this operation. He described his method in 1848.

Cannaday[76] reemphasizes the importance of the use of cutis in surgical procedures. He thinks that it is an ideal material for ligation of the common carotid artery and other large vessels.

(Comment.—The tissue skin is composed of two structures: the epidermis and the dermis. Why does one always call the outer layer the epidermis but change the term indicating the deeper layer according to fancy? If the outer layer is the epidermis, the deep layer should be called the dermis. If the deep layer is to be called cutis, why not be consistent and call the outer layer epicutis? [With apology to Dr. Cannaday, whom we admire.])

Barker,[77] of Philadelphia, describes a technic for injecting saline solution to fill out depressed areas so that the dermatome can be used to remove split thickness grafts from areas that would otherwise be useless.

In working with guinea pigs, Barker[78] showed that when an unpigmented area of skin is transplanted to a pigmented area, the pigment invades the unpigmented skin, obliterating it. When a pigmented area is transplanted to an unpigmented area, the pigment invades the surrounding tissue, increasing by several times the size of the pigmented area. In the transplanted unpigmented pieces, the color of hair remains the same longer than that of the skin. Also, the direction of the hairs remains the same throughout. This work confirms the results of the earlier investigators in this field.

74. Brown, J. B., and Cannon, B.: Composite Free Grafts of Two Surfaces of Skin and Cartilage from the Ear, Ann. Surg. **124**:1101 (Dec.) 1946.

75. Fogh-Anderson, P.: Stein-Estlander-Abbe Operation, Plast. & Reconstruct. Surg. **3**:186 (March) 1948.

76. Cannaday, J. E.: Cutis: Its Various Uses in Surgery, J. Internat. Coll. Surgeons **11**:282 (May-June) 1948.

77. Barker, D. E.: New Donor Areas in Skin Grafting, Ann. Surg. **127**:410 (March) 1948.

78. Barker, D. E.: Pigment Changes in Experimental Whole Thickness Skin Grafts, Arch. Path. **44**:163 (Aug.) 1947.

HEMANGIOMAS

The article by Figi [79] on the treatment of hemangiomas of the head and neck is excellently presented whether one agrees with his ideas or not. It has been estimated that 95 per cent of hemangiomas appear in these locations. Following is a brief summary of Figi's article.

Capillary Hemangiomas.—Strawberry birthmark, or hemangioma simplex, lined with embryonic epithelium, is usually best dealt with by means of a screened radium plaque. Port wine stains (hemangioma congenitale), formed largely of blood vessels lined with adult epithelium, should not be treated with radiotherapy. If any treatment is necessary, the lesions should be excised and a skin graft applied.

Cavernous Hemangiomas.—These hemangiomas are soft compressible tumors of large thin-walled sinuses. In infants and children, these lesions are treated with radiation. (Figi perfers radon seeds.) In adults, the lesion may be treated with sclerosing agents, but the author finds the use of electrocoagulation more satisfactory.

Plexiform or Racemose Hemangiomas.—These lesions probably are essentially cavernous hemangiomas with single or multiple arteriovenous fistulas. They occur oftenest in adults. Electrocoagulation may be used. If the lesion is not too extensive it may be excised and the afferent vessels tied.

Phleboliths.—If these lesions cause discomfort they should be removed.

According to Owens and Stephenson,[80] 56 per cent of hemangiomas occur on the head and neck. These workers limit themselves to surgical excision and injection of sodium morrhuate. They have completely discarded the use of carbon dioxide snow and fear the dangers of any form of radiation therapy.

The authors use surgical excision when there is no response to injection or when hemorrhage, malignant degeneration or cirsoid aneurysm is present.

TATTOOING AND SANDPAPERING

Conway and Docktor [81] state the advantages of the tattoo method of treating capillary hemangiomas of the face over other methods now in use and present a detailed account of their technic in the treatment of

79. Figi, F. A.: Treatment of Hemangiomas of the Head and Neck, Plast. & Reconstruct. Surg. **3**:1 (Jan.) 1948.

80. Owens, N., and Stephenson, K. L.: Hemangioma: An Evaluation of Treatment by Injection and Surgery, Plast. & Reconstruct. Surg. **3**:109 (March) 1948.

81. Conway, H., and Docktor, J. P.: Neutralization of Color in Capillary Hemangiomas of the Face by Intradermal Injection (Tattooing) of Permanent Pigments, Surg., Gynec. & Obst. **84**:866 (April 15) 1947.

seven patients. In all the completed cases, marked improvement was shown. The pigment must be injected into the derma, deep to the stratum germinativum of the epidermis but superficial to the vascular dilatations of the tumor. In adults, no anesthesia is necessary. Injections are given at intervals of two weeks and cover areas of 2 to 3 square inches (13 to 19 sq. cm.) at a time.

The removal of traumatic tattoos with sandpaper was originally devised, according to Iverson,[82] during World War II, the trauma being due to the explosion of land mines. This treatment is based on the finding that traumatic tattoos for the most part do not penetrate beneath the dermal layer of the skin. The commercial tattoo is deeper and is not amenable to this method. Iverson's technic is as follows. First,

Fig. 6.—*A*, patient injured January 1947 in iron foundry by impregnation of oil and dirt by high compression hose. *B*, six weeks postoperatively. A few remaining flecks in the subcutaneous tissue have since been removed by simple excision. (From Iverson.[82])

prepare a few gauze rolls by covering with small pieces of ordinary sandpaper, grit 0 and 00, and autoclaving. Anesthetize the area by local infiltration with a solution of procaine and epinephrine and abrade vigorously with the sandpaper roll. Wash off the detached particles of sand with saline solution and dry the area with gentle gauze pressure.

(*Comment.*—The results obtained, as demonstrated in the illustrations [fig. 6] would appear to bear out the merit of Iverson's technic, which seems to be an important practical contribution.)

82. Iverson, P. C.: Surgical Removal of Traumatic Tattoos of the Face, Plast. & Reconstruct. Surg. **2**:427 (Sept.) 1947.

A method for the permanent pigment injection of corneal scars is described by Pickrell and Clark.[83] The result may be beneficial not only cosmetically but also by the improvement of visual acuity through the absorption of certain peripheral rays of light which cause spherical aberration. A number of excellent photographs are included.

DEVELOPMENTAL ODDITIES

The condition of a small lower jaw, known as micrognathia, is discussed by Douglas,[84] with emphasis on the danger of the associated respiratory dyspnea. Such dyspnea is caused by a "linguo-epiglottic obstruction" due to the backward position of the tongue. It can be corrected by an operation in which the tongue is held forward by fusing it to the lower lip. Douglas describes his operation and reports six cases.

In pseudoxanthoma elasticum, as stated by Pickrell and his associates,[85] cutaneous and retinal manifestations may occur separately or simultaneously. The retina presents angioid streaks and may be associated with varying degrees of choroiditis. In the case reported here, the cutaneous manifestations were a purposeless reduplication of skin folds of the neck and axillas. A plausible explanation is that both conditions result from degenerative changes in the elastic tissue.

Because there is so little embryologic and histological distinction between dermoids and epidermoids, Pfeiffer and Nicholl[86] contend that the name "dermoid" is sufficient to cover both. A series of cases is presented by the authors primarily to call attention to defects in the bone which the lesions produced. The defects may be well studied by roentgenography, which serves to distinguish these lesions from other causes of proptosis. The proper use of phenolization is mentioned, and surgical approaches are described.

As pointed out by Kostrubala and Talbot,[87] myositis ossificans of the masseter muscle is rare. There is a tendency to recurrence of calcification when a bony mass is removed, probably because of the presence of dead space. The authors recommend obliterating this space with a dermal graft.

83. Pickrell, K. J., and Clark, E. H.: Tattooing of Corneal Scars with Insoluble Pigments, Plast. & Reconstruct. Surg. **2**:44 (Jan.) 1947.

84. Douglas, B.: The Treatment of Micrognathia Associated with Obstruction by a Plastic Operation, Plast. & Reconstruct. Surg. **1**:300 (Nov.) 1946.

85. Pickrell, K. L.; Kelly, J. W., and Marzoni, F. A.: The Plastic Surgical Treatment of Pseudoxanthoma Elasticum, Plast. & Reconstruct. Surg. **3**:700 (Nov.) 1948.

86. Pfeiffer, R. L., and Nicholl, R. J.: Dermoids and Epidermoids of the Orbit, Tr. Am. Ophth. Soc. **46**:218, 1948.

87. Kostrubala, J. G., and Talbot, R. J.: Myositis Ossificans of the Masseter Muscle, Plast. & Reconstruct. Surg. **3**:52 (Jan.) 1948.

McLaughlin[88] reports a very interesting and rare case of reduplication of the mouth, tongue and mandible in an infant. These parts were surgically excised.

La Ferté[89] describes his method for the correction of congenital torticollis, emphasizing, in certain cases, the importance of cutting platysma muscle fibers on the affected side as well as the origins of the sternocleidomastoid muscle. He believes that a vertical incision is preferable to a horizontal one, since it permits earlier motion, both active and passive. Cotton pads instead of a plaster cast are used to maintain the head in an overcorrected position postoperatively.

As ascertained by Davis and Berner,[90] only 50 cases of dermoid cysts of the nose have been recorded in the medical literature. These lesions are of special interest because of the embryology. The roentgenograms and instillations of iodized oil (lipiodol®) are important in diagnosis. Dermoid cysts of the nose are frequently not recognized nor adequately removed.

Double lip, a developmental anomaly, is due to a redundancy of tissue in the mucosal portion of the lip just inside the vermilion border. When the patient smiles, this accessory fold of tissue appears as a second lip. Dingman[91] corrects the condition by simple excision of the excess tissue in a horizontal eliptical piece.

RECENT DEVICES

Douglas and Millikan[92] have devised a photoelectric method for testing the circulation of blood in pedicle flaps. This "oximeter test" is very rapid and is highly reliable in predicting successful transfer. The method may eventually have a wider surgical and medical use than it has at present.

An obturator for use in feeding patients with cleft palate is described by Albray.[93] The obturator is made of plastic (lucitone®) and is attached to the bottle at the base of the nipple by stainless steel wires.

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88. McLaughlin, C. R.: Reduplication of Mouth, Tongue and Mandible, Brit. J. Plast. Surg. **1**:89 (July) 1948.

89. LaFerté, A. D.: The Role of the Platysma Muscle in Torticollis Deformity, Plast. & Reconstruct. Surg. **2**:72 (Jan.) 1947.

90. Davis, A. D., and Berner, R. E.: Dermoid Cysts of the Nose, Plast. & Reconstruct. Surg. **3**:345 (May) 1948.

91. Dingman, R. O., and Billman, H.: Double Lip, J. Oral Surg. **5**:146 (April) 1947.

92. Douglas, B., and Millikan, G. A.: The Blood Circulation in Pedicle Flaps: Preliminary Studies on a Photo-Electric Test for Determining Its Efficiency, Plast. & Reconstruct. Surg. **2**:348 (July) 1947.

93. Albray, R. A.: An Obturator for Use in Feeding Cleft Palate Cases, Plast. & Reconstruct. Surg. **2**:281 (May) 1947.

Martin [94] describes a tapered, angled tube that holds the tracheal stump out of the way so that no blood enters the tracheobronchial tree after the trachea has been transected just below the cricoid cartilage.

The "chondrojet" is a new instrument devised by De Kleine.[95] It is very neat and compact for handling diced cartilage.

González Ulloa [96] describes a splinting mechanism for correcting compound fractures. This mechanism is for use in comminuted fractures with considerable displacement, particularly when posterior displacement is likely. The use of this gadget does not interfere with normal breathing.

In the January issue of "Plastic and Reconstructive Surgery," this same author presents another device called the "dermo-coaptor," which has two hooks on adjustable bolts attached to a bar. This mechanism is used to exert outward tension at both ends of the wound during suturing and may be of value when the surgeon has no assistant.[97]

A device called a "vacutome" is introduced by Barber,[98] of Philadelphia. It appears to have definite advantages over the dermatome.

Macmillan [99] describes a point-eyed suture set that appears to be extremely simple in operation and delicate enough to be used in suturing the eye or for harelip and cleft palate work. It uses easily replaceable needles. The author has also devised a multiple knife for dicing cartilage.[100]

Aluminum foil as a dry sterile initial covering for thermal burns under occlusion pressure is presented by Brown, Farmer and Franks [101] (of Toronto) as a method for diminishing the maceration of a burn surface. The method appeared to exert a favorable influence on the local result by elimination of the use of ointments and by facilitating

94. Martin, H.: Tracheal Airway for Use During Total Laryngectomy, Am. J. Surg. **75**:755 (July) 1948.

95. De Kleine, E. H.: The Chondrojet: Simplified Method for Handling of Diced Cartilage, Plast. & Reconstruct. Surg. **3**:95 (Jan.) 1948.

96. González Ulloa, M.: Nose Fractures Treated with the Rhinotractor, Plast. & Reconstruct. Surg. **2**:607 (Nov.) 1947.

97. González Ulloa, M.: A New Device for Easier Skin Suturing, Plast. & Reconstruct. Surg. **3**:92 (Jan.) 1948.

98. Barker, D. E.: Vacutome—New Machine for Obtaining Split Thickness Skin Grafts, Plast. & Reconstruct. Surg. **3**:492 (July) 1948.

99. Macmillan, W.: A Point-Eyed Suture Set, Plast. & Reconstruct. Surg. **3**:222 (March) 1948.

100. Macmillan, W.: A Multiple Knife for Dicing Cartilage, Plast. & Reconstruct. Surg. **3**:226 (March) 1948.

101. Brown, W. A.; Farmer, A. W., and Franks, W. R.: Local Application of Aluminum Foil and Other Substances in Burn Therapy, Am. J. Surg. **76**:594 (Nov.) 1948.

dispersion of exudate to the periphery of the burn. No evidence of toxicity was found as a result of the treatment. The study was based on a series of 50 cases.

Penn,[102] of South Africa, states that for two years he has been using grated cadaver cartilage as a contour former. He presents a cylinder for this use made for him by Dr. Robert Ivy.

BLOOD STUDY

One of the most important contributions to all types of surgical procedure was the observations made by Beling, Morton and Bosch[103] in their study of blood volume in preoperative and postoperative care.

They found that many patients with a variety of diseases have shrunken total blood volume with deficits of plasma, hemoglobin and cells, circulating plasma proteins and body proteins. The decreased blood volume with accompanying deficits may not be disclosed by the patient's weight and the usual laboratory estimations of hemoglobin, hematocrit and plasma proteins. If the decreased blood volume and its associated deficits are not recognized, there may be serious consequences for the patient during and after operation.

A simple routine is proposed which is practical for any small hospital and which may give accurate appraisal of a patient's status before operation.

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102. Penn, J.; Jonkowitz, J., and Bruwer, A.: A Note on the Use of Grated Cadaver Cartilage, Plast. & Reconstruct. Surg. **3**:228 (March) 1948.

103. Beling, C. A.; Morton, T. V., and Bosch, D. T.: Blood Volume and Other Determinations in Preoperative and Postoperative Care, Surg., Gynec. & Obst. **87**:163 (Aug.) 1948.

Special Reports

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE AND REHABILITATION, THE AMERICAN MEDICAL ASSOCIATION

As of November 1, 1950

Aurex Hearing Aid Model F	J. A. M. A. **138**:294 (Sept. 25) 1948.
Aurex Hearing Aid Model H Mfr. Aurex Corporation 1117 North Franklin Street Chicago 10	J. A. M. A. **136**:1099 (April 24) 1948.
Beltone Mono-Pac Hearing Aid	J. A. M. A. **130**:637 (March 9) 1946.
Beltone Harmony Mono-Pac Hearing Aid	J. A. M. A. **133**:543 (Feb. 22) 1947.
Beltone Symphonette	J. A. M. A. **140**:1217 (Aug. 13) 1949.
Beltone Mono-Pac Model M Hearing Aid Mfr. Beltone Hearing Aid Company 1450 West Nineteenth Street Chicago 8	J. A. M. A. **144**:929 (Nov. 11) 1950
Cleartone Hearing Aid Model 500	J. A. M. A. **144**:103 (Sept. 9) 1950.
Cleartone Hearing Aid Regency Model Mfr. American Sound Products, Inc. 2454 South Michigan Avenue Chicago 16	J. A. M. A. **144**:103 (Sept. 9) 1950.
Dysonic Hearing Aid Model 1 Mfr. Dynamic Hearing Aids, Inc. 1042 Atlantic Avenue Brooklyn 16	J. A. M. A. **137**:1534 (Aug. 21) 1948.
Electro-Ear Hearing Aid Model C Mfr. American Earphone Co., Inc. 10 East Forty-Third Street New York 17	J. A. M. A. **136**:769 (March 13) 1948.
Gem Hearing Aid Model V-35	J. A. M. A. **139**:229 (Jan. 22) 1949.
Gem Hearing Aid Model V-60 Mfr. The Gem Ear Phone Co., Inc. 50 West Twenty-Ninth Street New York 1	J. A. M. A. **144**:847 (Nov. 4) 1950.
Maico Atomeer Hearing Aid	J. A. M. A. **133**:542 (Feb. 22) 1947.
Maico UE Atomeer Hearing Aid	J. A. M. A. **143**:1069 (July 22) 1950.
Maico Quiet Ear Models G and H Mfr. Maico Company, Inc. 21 North Third Street Minneapolis 1	Report not yet published.

Mears Aurophone Hearing Aid Model 200 J. A. M. A. **138**:428 (Oct. 9) 1948.
1947 Mears Aurophone Model 98 J. A. M. A. **137**:1535 (Aug. 21) 1948.
Mfr. Mears Radio Hearing Device Corp.
1 West Thirty-Fourth Street
New York 1

Micronic Hearing Aid Model 101 (Magnetic Receiver) J. A. M. A. **139**:99 (Jan. 8) 1949.
Micronic Hearing Aid Model 303 J. A. M. A. **142**:1.63 (April 29) 1950.
Mfr. Micronic Company
727 Atlantic Avenue
Boston 11
(See Silver Micronic)

Microtone Audiomatic T-3 Hearing Aid J. A. M. A. **138**:1229 (Dec. 25) 1948.
Microtone Audiomatic Hearing Aid Model T-4 J. A. M. A. **136**:109 (Jan. 10) 1948.
Microtone Audiomatic Model T-5 Hearing Aid J. A. M. A. **139**:379 (Feb. 5) 1949.
Microtone Classic Model T-9 Report not yet published.
Microtone Model 45 J. A. M. A. **144**:928 (Nov. 11) 1950.
Mfr. The Microtone Company
Ford Parkway on Mississippi
St. Paul 1

National Hearing Aid Cub Model (C) J. A. M. A. **138**:295 (Sept. 25) 1948.
National Standard Model (T) J. A. M. A. **138**:295 (Sept. 25) 1948.
National Star Model (S) J. A. M. A. **138**:295 (Sept. 25) 1948.
National Ultrathin Model 504 Report not yet published.
National Vanity Model 506 Report not yet published.
Mfr. National Hearing Aid Laboratories
815 South Hill Street
Los Angeles 14

Otarion Hearing Aid Model E-1 J. A. M. A. **136**:108 (Jan. 10) 1948.
Otarion Hearing Aid Model E-1 S J. A. M. A. **138**:650 (Oct. 30) 1948.
Otarion Hearing Aid Model E-2 J. A. M. A. **138**:887 (Nov. 20) 1948.
Otarion Hearing Aid Model E-4 J. A. M. A. **140**:156 (May 14) 1949.
Otarion Hearing Aid Models F-1 and F-2 J. A. M. A. **144**:762 (Oct. 28) 1950.
Mfr. Otarion, Inc.
159 North Dearborn Street
Chicago 1

Paravox Hearing Aids Models VH and VL (Standard) J. A. M. A. **132**:79 (Sept. 14) 1946.
Paravox Hearing Aid Model XT (Extra thin) J. A. M. A. **134**:365 (May 24) 1947.
Paravox Hearing Aid Model XTS (Extra thin) J. A. M. A. **136**:109 (Jan. 10) 1948.

Paravox Hearing Aid Model Y (YM., YC and YC-7) (Veri-small) J. A. M. A. **140**:155 (May 14) 1949.
Mfr. Paravox, Inc.
2056 East Fourth Street
Cleveland 15

Radio Permo-Magnetic Hearing Aid (Multipower Model) J. A. M. A. **140**:959 (July 16) 1949.

Radiocar Permo-Magnetic Hearing Aid (Uniphone One-Piece Model) J. A. M. A. **139**:787 (March 19) 1949.

Radiocar All-Magnetic Model 55 Hearing Aid J. A. M. A. **143**:555 (June 10) 1950.

Radiocar Model 62 Starlet J. A. M. A. **144**:929 (Nov. 11) 1950.
Mfr. E. A. Myers & Sons
306-308 Beverly Road
Mt. Lebanon
Pittsburgh 16

Silver Micronic Hearing Aids (Magnetic and Crystal) Models 202 M and 202 C J. A. M. A. **139**:848 (March 26) 1949.
Mfr. Micronic Company
727 Atlantic Avenue
Boston 11
(See Micronic)

Silvertone Hearing Aid Model 103 BM J. A. M. A. **142**:815 (March 18) 1950
Mfr. National Hearing Aid Laboratories
815 South Hill Street
Los Angeles 14
Distr. Sears, Roebuck & Company
925 South Homan Ave
Chicago 7

Silvertone Model M-35 Report not yet published.
Mfr. Micronic Company
727 Atlantic Avenue
Boston 11
Distr. Sears, Roebuck & Company
925 South Homan Ave
Chicago 7

Silvertone Model P-15 Report not yet published.
Mfr. W. E. Johnston Mfg. Company
708 West Fortieth Street
Minneapolis
Distr. Sears, Roebuck & Company
925 South Homan Avenue
Chicago 7

Solo-Pak Model 99 Hearing Aid J. A. M. A. **144**:315 (Sept. 23) 1950.
Mfr. Solo-Pak Electronics Corp.
Linden Street
Reading, Mass.

Sonotone Vacuum Tube Hearing Aid Model 600	J. A. M. A. **131**:523 (June 8) 1946.
Sonotone Hearing Aid Model 700	J. A. M. A. **135**:838 (Nov. 29) 1947.
Sonotone Hearing Aid Model 900	J. A. M. A. **139**:786 (March 19) 1949.
Sonotone Hearing Aids Models 910 and 920	J. A. M. A. **142**:815 (March 18) 1950.
Sonotone Hearing Aid Model 925 Mfr. Sonotone Corporation Elmsford, N. Y.	J. A. M. A. **144**:315 (Sept. 23) 1950.
Super-Fonic Hearing Aid Mfr. American Sound Products, Inc. 2454 South Michigan Avenue Chicago 16	J. A. M. A. **138**:293 (Sept. 25) 1948.
Televox Model E Hearing Aid Mfr. Televox Manufacturing Company 1307 Sansom Street Philadelphia 7	J. A. M. A. **140**:783 (July 2) 1949.
Telex Hearing Aid Model 22	J. A. M. A. **134**:605 (June 14) 1947.
Telex Hearing Aid Model 97	J. A. M. A. **138**:294 (Sept. 25) 1948.
Telex Hearing Aid Model 99	J. A. M. A. **141**:1153 (Dec. 17) 1949.
Telex Hearing Aid Model 200	J. A. M. A. **144**:103 (Sept. 9) 1950.
Telex Hearing Aid Model 1700 Mfr. Telex, Inc. Telex Park Minneapolis 1	J. A. M. A. **141**:1057 (Dec. 10) 1949.
Tonemaster Model Royal Hearing Aid Mfr. Tonemaster, Inc. 400 South Washington Street Peoria 2, Ill.	J. A. M. A. **139**:848 (March 26) 1949.
Trimm Vacuum Tube Hearing Aid Model 300 Mfr. Trimm Distributors, Inc. 400 Lake Street (P. O. Box 489) Libertyville, Ill.	J. A. M. A. **133**:542 (Feb. 22) 1947.
Unex Hearing Aid Model A	J. A. M. A. **134**:254 (May 17) 1947.
Unex Midget Model 95	J. A. M. A. **141**:134 (Sept. 10) 1949.
Unex Midget Model 110 Mfr. Nichols and Clark Hathorne, Mass.	J. A. M. A. **141**:134 (Sept. 10) 1949.
Vacolite Model J Hearing Aid Mfr. Vacolite Company 3003 North Henderson Street Dallas 6, Texas	J. A. M. A. **140**:782 (July 2) 1949.

Western Electric Hearing Aid Model 63 — J. A. M. A. **131**:895 (July 13) 1946.

Western Electric Hearing Aid Model 64 — J. A. M. A. **134**:605 (June 14) 1947.

Western Electric Hearing Aids Models 65 and 66 — J. A. M. A. **137**:534 (June 5) 1948.

Mfr. Western Electric Company, Inc.
120 Broadway
New York 5

Zenith Hearing Aid Model 75 — J. A. M. A. **135**:773 (Nov. 22) 1947.

Zenith Miniature 75 Hearing Aid — J. A. M. A. **143**:440 (June 3) 1950

Mfr. Zenith Radio Corporation
6001 West Dickens Avenue
Chicago 39

(All of the accepted hearing devices employ vacuum tubes.)

Accepted hearing aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS

Aurex Hearing Aid (Semiportable) — J. A. M. A. **109**:585 (Aug. 21) 1937.

Mfr. Aurex Corporation
1117 North Franklin Street
Chicago 10

Precision Table Hearing Aid — J. A. M. A. **139**:785 (March 19) 1949.

Mfr. Precision Electronics Company
5157 West Grand Avenue
Chicago 39

Sonotone Professional Table Set Model 50 — J. A. M. A. **141**:658 (Nov. 5) 1949.

Mfr. Sonotone Corporation
Elmsford, N. Y.

Abstracts from Current Literature

Ear

THE TOXIC EFFECT OF STREPTOMYCIN ON THE EIGHTH CRANIAL NERVE: HISTOLOGICAL INVESTIGATIONS; A PRELIMINARY REPORT. KARL BERG, Ann. Otol., Rhin. & Laryng. **58**:448 (June) 1949.

In an effort to determine the actual site of the lesion responsible for vestibular dysfunction unassociated with hearing loss in patients receiving streptomycin over prolonged periods, the author administered the drug twice daily in varying amounts to 25 young cats. The total dose for any animal was the equivalent of 156 to 965 Gm. for an adult weighing 70 Kg. The duration of treatment ranged from 14 to 127 days. Vestibular function was tested regularly by the rotatory test, the postrotatory nystagmus being recorded. Ultimately, nystagmus became extinct, the time required varying according to the total amount of drug administered.

On conclusion of the experiment, all animals except three were killed, and the vestibular ganglion, basal coil of the cochlea and the vestibule were studied histologically. Studies on the remaining three animals will form the basis for a further report. Marked degenerative changes in the sensory epithelium of the crista ampullaris, macula utriculi and macula sacculi were noted in each animal. The organ of Corti was normal in all except one animal, which had received the largest dose of streptomycin (roughly equivalent to 960 Gm. in man, over a period of 127 days). In this instance, considerable degeneration of the sensory epithelium in the organ of Corti was found.

Study of the brain and vestibular nuclei showed no definite pathologic changes. Slight changes in the Nissl substance of the ganglion cells were present.

It is concluded that streptomycin affects primarily the sensory epithelium of the labyrinth with slight secondary changes in the vestibular ganglion.

STEELE, Kansas City, Kan.

INTERPRETATION OF HEARING TESTS. STACY R. GUILD, J. A. M. A. **142**:466 (Feb. 18) 1950.

The author briefly reviews his conclusions in a paper of the same title presented the previous year.

This article is concerned with the problem of conduction deafness, and by means of schemata and charts the author discusses moderately the routes of conduction deafness. He points out that a far greater amount of acoustic energy is required to produce a sensation of hearing by bone conduction than is normally required for hearing by air conduction and that the possible presence of a conductive lesion is not ruled out of consideration when the answer to the qualitative test is that the fork sounds louder by air conduction than by bone conduction. It is important to time the duration of hearing by each route as well as to know what the normal times of the particular fork may be. Suitable masking should be used during the testing.

Impairment may be approximately equal for all tones for air conduction or may be greater for low tones than for high ones and vice versa.

That impairment for high tones in air conduction testing has been found in cases of conduction deafness has been proved and is accepted. That impairment for high tones has been found in cases of simple conduction deafness, as by occlusion of the external auditory canal with either cerumen or an ear plug, should make the clinical otologist discard the classic concept still repeated in textbooks that a greater loss of hearing for high tones than for low tones always means nerve deafness. GORDON, Philadelphia.

OTOLARYNGOLOGICAL EXPERIENCES IN A HEARING SURVEY. ARNOLD GROSSMAN and RICHARD E. MARCUS, J. Speech & Hearing Disorders **14**:240, 1949.

In Will County, Ill., a hearing program was started on Nov. 3, 1947, and all school children through the ages of 6 to 19 were tested during the following seven months. There were 20,663 children, and during the first visit 4.7 per cent were absent; 581 of the 971 originally absent were recorded later, leaving an absentee record of 1.8 per cent. There were 1,646 children, or 7.9 per cent, who required otological investigation on the basis of a 30 decibel loss in either ear and at any frequency between 128 and 8,192 cycles per second. Males far outnumbered females.

Only 0.4 per cent of the referred subjects were found to be normal on clinical examination; 1,471 children were examined by a program otologist in the presence of the parents. In 12 per cent of the cases a clinical syndrome of allergic rhinosalpingitis seemed suggestive as a cause of hearing loss.

Continuous follow-up observation was maintained from the moment of screening through the otological clinic and through the referral to family physicians or otolaryngologists of choice. Diagnosis, recommendations and treatments were recorded. Educative and rehabilitative follow-up observations were maintained.

PALMER, Wichita, Kan.

AUDITION AND PHONATION. EDUARDO CASTERAN, Rev. de argent. oto-rino-laring. 1949.

At the first Latin American Congress of Otorinolaryngology, held in Santiago, Chile, in January 1949, Casteran presented a monograph on audition and phonation. This work is an extensive detailed discussion of the many problems. The first chapter deals with the ear and the spoken voice and includes the role of the receptive centers for speech and hearing and some of the psychosomatic problems associated with deaf-mutism. The second chapter is concerned with acoustic disturbances in aphasic syndromes. The chapter deals with disturbances of phonation and the fourth chapter with deaf-mutism. The fifth chapter is concerned with audition and phonation among school children. In the sixth chapter he discusses audiometry. Casteran devoted the entire last chapter to presenting a comprehensive plan for establishment of hearing centers from the standpoint of prophylaxis and therapy. He advocates federal control to establish centers in which early diagnosis of hearing defects can be made, not only among school children but even among the preschool group. He suggests further that these children be sent to special schools where they can obtain proper training and education. These schools should also be directly integrated with teaching institutions so that not only the local problems can be evaluated but a unified survey plus considerable propaganda in the forms of various educational programs should be developed. PERSKY, Philadelphia.

SIGNIFICANCE OF THE FREQUENCY IN CALORIC NYSTAGMUS. NICHOLAS TOROK, Acta oto-laryng. **36**:40, 1948.

The author adds a new concept to the already established time concept of the latency period and duration of the nystagmus period in caloric testing. He states that the number of vibrations per unit of time are important. The nystagmus frequency changes during the duration of the nystagmus reaction and can be plotted as a curve. It is important, therefore, that the frequency be determined in all instances during the same period of time relative to the time of onset of the nystagmus.

The counting of the nystagmus therefore is established for a 10 second period between the twenty-fifth and thirty-fifth second after placing the head in the optimum position.

He establishes the physiologic value of the nystagmus in these circumstances as between 14 to 18 vibrations. His studies indicate that there can be absolute and relative deviations between nystagmus duration and frequency.

Nystagmus frequency is the ratio between the slow and the quick component. Variations from the standard may lead to a new realization of pathologic states and may eventually result in help in cerebral localization in the central parietal area of the cortex of the side opposite to the ear tested.

ROBERT LEWY, Chicago.

THE PASSAGE OF FLUORESCEIN SODIUM TO THE LABYRINTHINE FLUIDS. LENNART GISSELSSON, Acta oto-laryng. **37**: 268 (June) 1949.

These experiments were set up because of the slight existing knowledge of the source, flow and ultimate fate of the labyrinthine fluids. The work was done on guinea pigs and cats. It involved making an opening into the apex of the cochlea, injecting the drug fluorescein intra-arterially and then, after a period of time, drawing off perilymph and studying this under ultraviolet light for the fluorescein. In cats the capillary tube was placed in the round window.

The experiments showed that fluorescein passed from the blood into the cerebrospinal fluid in a few minutes. The spread into the perilymph was, however, not demonstrable until several hours later. The exception to this was the presence of a pathologic condition when trauma created a leak. In the cat, the dye passed into the perilymph as rapidly as it did into the cerebrospinal fluid. The results suggest that endolymph is formed by secretion in the membranous labyrinth, and perilymph is derived mainly from the cerebrospinal fluid.

ROBERT LEWY, Chicago.

SURGICAL TREATMENT OF CHRONIC MIDDLE EAR INFLAMMATION WITH CENTRAL PERFORATION OF THE DRUM. KARL BERG and TRYGVE FALKENBERG, Acta oto-laryng. **37**:415 (Oct.) 1949.

The purpose of this article was to distinguish a certain group of cases of chronic otitis, those in which continuous secretion and central perforation were presented, and to try to demonstrate that cure may be effected in these cases by surgical treatment. The authors state that continuous discharge is an indication of changes in the mastoid bone, and in those instances in which the discharge is periodic the pathologic condition is localized to the mucous membrane.

The material consisted of 15 ears with the characteristics described above. In the group of ears that were operated on, discharge had been going on from two to 28 years. All these ears were dry on an average of the seventeenth postoperative day. Five of the control ears also became dry in a corresponding period. The degree of pneumatization, studied roentgenographically by three different views of the mastoid, did not seem to have any bearing on the postoperative result.

The results could not be considered lasting. Only five ears were dry 8, 9, 11 and 12 months after the operation. Six ears showed improvement and three showed no improvement.

ROBERT LEWY, Chicago.

SURGICAL TREATMENT OF HEADACHE SECONDARY TO EAR SUPPURATION. A. AUBIN, Acta oto-rhino-laryng. belg. **2**:345, 1948.

A group of cases is reported of unilateral headaches in the temporal and parietal area secondary to radical mastoidectomy. These headaches did not respond to sedatives. The eyegrounds were normal, no symptoms of increased intracranial pressure were present and the results of vestibular tests were normal, but a sensation of vertigo and tinnitus occurred without exact localization. The skull trepanation showed a herniating dura, a hypervascularized brain and a jelly-like substance filling the subarachnoid space.

The usual decompression operations did not relieve the headache. Inspired by the works of Penfield, de Seze and Guillaume on the meningeal vascularization and circulation, the author carried out removal of a section of the ophthalmic branch of the trigeminal nerve, with resulting anesthesia of the involved meninges and cessation of headaches. He states that the following branches are involved in postoperative otogenous headaches: the ethmoidal branch of the nasal nerve, distributed to the dura of the frontal region; Arnold's recurrent nerve, distributed to the occipital dura (tentorium cerebelli), and a branch of the mandibular nerve (otic ganglion), forming a plexus around the middle meningeal artery and distributing sensibility to the parietal and temporal dura.

HERSON, Chicago.

ROENTGENOLOGIC TECHNIC AND ANALYSIS OF THE DISEASES OF THE PETROMASTOID STRUCTURE. R. GILBERT and G. VOLUTER, Pract. oto-rhino-laryng. **11**:92, 1949.

The authors discuss the usual technics used in examination of the petromastoid structure, such as those employed in the Viennese school, or by Stenvers in Holland or by Chausse in Paris. They evaluate the advantages and disadvantages of each method, and, in consequence, they use a bilateral projection, symmetrical to the fronto-occipital plane, regulating this in such a manner that they are able to analyze at one time without any loss of orientation the pathologic modifications of the mastoid cells extending in the petrous pyramid.

Their method is to flex the head forward so that the chin almost rests on the Adam's apple. The roentgen ray tube is centered on the frontal bone, forming an angle of about 60 degrees with the horizontal auricular plane and passing through the occiput; the rays would emerge toward the summit of the neck in the subocciptal region. This method presents all bony anomalies, fractures of the base of the skull and middle ear infections plus their complications, such as mastoiditis and cholesteatoma. Bony modifications of the internal surfaces and the tip of the petrous are clearly visualized.

PERSKY, Philadelphia.

Larynx

HISTOPLASMOSIS OF THE LARYNX. EDWIN D. GAMMELL and ROBERT L. BRECKENRIDGE, Ann. Otol., Rhin. & Laryng. **58**:249 (March) 1949.

Gammell and Breckenridge add two cases of laryngeal histoplasmosis to those previously reported by other authors, bringing the total of reported cases to 12.

The two cases were similar in a number of respects. Hoarseness was the chief complaint, and the laryngeal lesions were responsible for the patients' having been admitted to the hospital. Laryngoscopy showed an ulcerated, granular, infiltrating lesion involving the vocal cords and extending into the ventricles. Fixation of the involved vocal cords was present. Biopsy revealed granulomatous lesions containing the yeast form of Histoplasma capsulatum within macrophages and monocytes.

Difficulty in isolating the organism on artificial media was experienced in both cases. Seeding on Sabouraud's glucose agar kept at room temperature for at least one month was found to be the most satisfactory technic. The results of histoplasmin skin test were either negative or weakly positive. On necropsy, adrenal gland involvement was found in both patients. While present day antibiotics were unable to combat the infection, it was believed that they may have been responsible for the disappearance of the laryngeal lesions in one of the cases.

STEELE, Kansas City, Kan.

TUBERCULOUS LARYNGITIS TREATED BY STREPTOMYCIN. WILLIAM MCKENZIE, J. Laryng. & Otol. **64**:167 (April) 1950.

This is a report of treatment with streptomycin of 12 patients with tuberculosis of the larynx. All were sanitarium patients and were treated only if a month's silence and routine care did not improve the larynx. One gram of streptomycin was given daily for three to four weeks. Six patients were cured; five were improved, and there was no change in one.

LEJEUNE AND LEWIS, New Orleans.

RENDU-OSLER DISEASE. G. MALFATTI, Oto-rino-laring. ital. **18**: 1949.

This article is a report of two cases, one of which is particularly interesting for the diagnostic difficulties presented. In fact, the telangiectasis in the case was localized 1 cm. from the bifurcation of the left bronchus, giving rise to hemoptysis not accounted for in spite of numerous physical and roentgenologic examinations. The results of the hemochromocytometric examination were within normal limits. Bronchoscopy finally revealed the lesion. After three months this condition was followed by similar neoformations on the tongue and the back. After the electrocoagulation of the dilated outstanding vessels, the cause of the hemorrhage, complete recovery was obtained. A strict interrogatory as to other cases of hemoptysis or epistaxis in the family revealed at least six cases of recurrent epistaxis. One of these was the other case presented, that of a 13 year old female child in whom the familial hemorrhagic angiomatosis was represented by telangiectasis of the locus Valsalvae.

As to therapy, electrocoagulation has been found to be better than radium, sclerosing injections and cauterizations.

MELUCCI, Philadelphia.

Nose

THE COLUMELLA. H. GILLES, Brit. J. of Plast. Surg. **2**:192 (Oct.) 1949.

Gillies' article is devoted to the various types of grafts using skin, cartilage and bone from various sources and the choice under different conditions. The chief addition to methods that are already familiar is the use of free heterografts, such as ox cartilage, to support the nasal bridge. A multistage procedure is recommended for many reconstructions.

SELTZER, Philadelphia.

TOMOGRAPHY OF THE PARANASAL SINUSES. R. REYNAUD and S. CABANETTES, Rev. de laryng. **70**:490 (Nov.) 1949.

The authors make roentgenographic plates of the paranasal sinuses on successive depths at 1 cm. intervals. At 1 cm. depth they visualize the frontal sinuses, the anterior part of the maxillary sinuses and the anterior ethmoidal cells; at 4 to 7 cm. the posterior ethmoidal cells and the posterior part of the antrum are in clear view, and at 7 to 8 cm. the sphenoid sinuses are. This concerns the fronto-occipital incidence.

The tomography in the anteroposterior plane (sagittal) is also very useful for visualization of the ethmoidal cells, sphenoidal sinuses and extension of the malignant tumors toward the posterior sinuses. The films are taken also at 1 cm. intervals. Oblique incidences at 45 degrees are also used by the authors for study of the extension of the sinuses and the relation between the optic foramens and the sphenoid sinuses.

HERSON, Chicago.

ACUTE FULMINATING OSTEOMYELITIS OF THE FRONTAL SINUS COMPLICATED BY MENINGITIS AND CEREBRAL ABSCESS. RUÁL VELASCO L., Rev. otorrinolaryng. **9**:57 (June) 1949.

The author presents an interesting case of a 17 year old youth in whom an acute frontal sinusitis developed after he went swimming while he had an acute infection of the upper respiratory tract. Within four days after the onset of his frontal symptoms, evidences of an osteomyelitis and meningitis appeared. Operation revealed a bilateral purulent frontal sinusitis with a frank osteomyelitis. There was an extensive involvement of the left half of the frontal bone and part of the parietal bone, with frank necrosis and the presence of a subdural abscess. In spite of operation and massive doses of penicillin, the temperature remained high, and within two days symptoms of a cerebral abscess were presented.

Although he was kept under observation for about three weeks, the symptoms still continued. The operative field was reviewed and an abscess found in the posterior part of the frontal lobe; 60 cc. of pus was removed. A week later another abscess in the parietal region was tapped with the removal of 40 cc. of fluid. Following the drainage of the second abscess the patient made an uneventful recovery.

The author points out certain important facts: 1. The infection was initiated by the patient's bathing in a swimming pool while having an acute infection of the upper respiratory tract. 2. Staphylococcus albus was the infecting organism. 3. A marked amount of spongy tissue was present throughout the bones of the skull. He also noted the surprising facts that the blood count and the spinal fluid were normal after the first operation, although two cerebral abscesses developed subsequently, and that massive doses of sulfadiazine, penicillin and streptomycin were unable to prevent the development and spread of the osteomyelitis and subsequently the brain abscesses but did prove effective in the control of the meningitis.

PERSKY, Philadelphia.

CONCERNING THE PATHOGENESIS OF THE CHOLESTEATOSIS OF THE MAXILLARY SINUS. G. B. PETRILLI, Valsalva **25**:136 (May-June) 1949.

After a brief discussion about the existing theories concerning the origin and development of cholesteatotic masses in the tympanic cavity and in the maxillary sinus, the author presents three cases in which the evolution and anamnesis show a definite causal relation between anomalies of dentition and cholesteatosis of the maxillary antrum.

The only situation that can explain the presence in the sinus of keratinizing epithelium is an ectopia of the epithelial element of the dental germ.

Early in its incipiency, the cholesteatotic tumor is nothing but a paradental cyst, which burrows its way into the sinus and eventually into the nasal chamber. At times symptoms are noticeable only at the latter stage.

Other proof of the likelihood of the author's statement as to the origin of the tumor is the fact that cholesteatosis has never been found in sinuses other than the maxillary.

MELUCCI, Philadelphia.

ON THE PRESENCE OF YEAST FUNGI IN THE CEREBROSPINAL FLUID IN OTO- AND RHINOGENIC MENINGITIDES. TAPIO SAVOLAINEN, Acta oto-laryng. **37**:339 (Aug.) 1949.

In 1861 the first published case of central nervous system disease by a fungus, Candida albicans (Oidium albicans), was presented by Zenker. At the University of Helsinki 16 patients with this condition have been observed in recent years.

All the patients had otogenic or rhinogenic disease. Thirteen of them suffered from purulent and two from serous meningitis. One of the group had normal cerebrospinal fluid except for the fungi. Yeast fungi cells were found to be present in 20 per cent of all recognized cases of meningitis. Culture of the yeast fungi was successful in two cases. One of the strains isolated was C. albicans (Monilia candida). C. albicans was also found in nasal cavities and ear canals of 23 per cent of 30 healthy women used as control cases. These strains were not, however, pathogenic to mice, but the strains cultured from the active cases were.

It is believed that the yeast fungi are of a secondary and saprophytic nature and that they arrive in the operative area through channels created by the operation. There were four cases in which the yeast was found in the cerebrospinal fluid preoperatively. In these instances they may have come in via the blood stream, the lymphatics or through olfactory channels.

ROBERT LEWY, Chicago.

THE SOURCE OF NASAL SECRETION IN THE NORMAL CONDITION: FLUORESCEIN TESTS. SVEN INGELSTEDT and BERTEL IVSTAM LUND, Acta oto-laryng. **37**:446 (Oct.) 1949.

In the studies reported here, 1 Gm. of sodium fluorescein, 5 ml. of a 20 per cent solution, was injected intravenously. At varying intervals thereafter, samples of nasal secretion were removed on cotton swabs from under the inferior turbinate body. The time allowed was usually thirty to forty-five minutes. The samples were then weighed, alkalinized with a 2 per cent solution of sodium bicarbonate and irradiated with a mercury vapor lamp. In none of 50 patients observed could fluorescein be demonstrated by this method.

The authors stated that these results could confirm the assumption that in the normal individual the nasal secretion is a pure glandular product.

ROBERT LEWY, Chicago.

THE SOURCE OF NASAL SECRETION IN INFECTIOUS, ALLERGIC AND EXPERIMENTAL CONDITIONS. SVEN INGELSTEDT and BERTEL IVSTAM LUND, Acta oto-laryng. **37**:451 (Oct.) 1949.

Intravenous fluorescein was administered, and the possible spread of this material to the nasal secretion in the non-normal situation of infection, allergy and experiment was studied. It was noted that fluorescein came through, and a bright fluorescence was observed as a general rule.

In chronic allergic rhinitis, passage of fluorescein to the secretion never occurred, this lack indicating the role of exudation in the formation of nasal secretion in infection and in acute allergic states. Antihistaminic drugs were able to inhibit completely the result when histamine was used experimentally.

ROBERT LEWY, Chicago.

Miscellaneous

SKIN SENSITIVITY TO STREPTOMYCIN. S. W. SIMON, J. Allergy **20**:56-61 (Jan.) 1949.

Three case histories are presented of women who acquired contact dermatitis from handling syringes containing streptomycin. These women worked in the central supply section of an institution in which approximately two hundred syringes were used daily in the administration of the drug. Reactions to patch testing with a 1 per cent solution of the drug was positive in each instance. Interestingly enough, no cases of contact dermatitis developed after the injection of the drug. The author concludes that sensitivity to streptomycin does exist and may develop in certain persons who have frequent contact with solutions of the drug and that past or present allergy has no effect on the appearance of this skin sensitivity. Routine patch testing of all personnel administering streptomycin or handling syringes which have contained it should be carried out every two months with a 1 per cent streptomycin solution. Effective prophylactic procedures are described.

FRIEDBERG, Chicago.

AUDIBILITY OF THE VOICELESS CONSONANTS AS A FUNCTION OF INTENSITY. GILBERT C. TOLHURST, J. Speech & Hearing Disorders **14**:210, 1949.

This article is a pilot study aimed at determining (1) the threshold of detectability of speech sounds or the point at which listeners would recognize a sound as being uttered without being able to identify it as a particular sound, and (2) the threshold or recognition of the phonetic unit itself. The sounds chosen for investigation were the nine surds, since these are closely connected with articulatory disorders. Nonsense syllables with the vowel "u" were utilized and presented as randomized intensities by two similar male voices to 22 observers having normal hearing as tested by pure tone audiometers.

Thresholds for voiceless consonant sounds were found at very low intensity levels. The data obtained from the two voices appeared to be similar. Detectability and recognition type judgments were found to yield similar thresholds for most of the sounds. Differences were small and varied. There was no real difference between the two types of recognition. The assumption that all sounds have audibility functions independent of their positions in syllables appeared unfounded. Sounds showing the greatest change due to position in the syllable were "k" and "p."

PALMER, Wichita, Kan.

NATURAL FREQUENCY, DURATION, AND INTENSITY OF VOWELS IN READING. JOHN W. BLACK, J. Speech & Hearing Disorders **14**:216, 1949.

In this study, 42 men read 11 words containing the various vowel sounds (1) after practice; (2) speaking at a 10 inch (25 cm.) distance from a microphone in a sound-treated room; (3) at five second intervals, viewing a 10 second sweep-hand timer, and (4) inhaling between each pair of words, the order of words being varied systematically.

Measurements were obtained of intensity, duration, frequency and the relations between these characteristics. There were no quantitative conclusions from this data, but independent analysis of various established significant differences between some vowels and natural frequencies, durations and intensities. Frequencies varied with the openness of the vowels; the more open the vowel, the lower the frequency. Duration varied directly with the openness of the vowels. There were a number of significant differences in over-all intensity between the vowels.

PALMER, Wichita, Kan.

VOICE RECORDING—SOME FINDINGS AND SOME PROBLEMS. ERNEST H. HENRIKSON and JOHN V. IRWIN, J. Speech & Hearing Disorders **14**:227, 1949.

In a speech clinic the recording process provides repeatable, preservable and independent stimuli. No direct benefit is derived from the presence of recording instruments. High fidelity equipment is a compromise among many factors but should have steady frequency responses not varying more than 5 decibels at any point on the curve between 80 to 8,000 cycles per second. Also, harmonic distortions and background noise should be at a certain optimum production level, and distortion on fifth harmonics should not exceed a critical percentage of 0.3. While this is only medium fidelity, probably only a few broadcasts of radio or reception exceed it or even approach it. Records, files, etc., involve considerable expense.

Durability is important since much fidelity apparatus requires electronic experts to keep it in operation. Students must have rugged recording instruments. The flexibility of the equipment purchase must also be considered, and, while there is a great amount of literature on the use of recording devices, very little experimentation has been done with objective technics evaluating the needs of the situation.

PALMER, Wichita, Kan.

HIGH CERVICAL ESOPHAGOGASTROSTOMY FOR CARCINOMA OF THE CERVICAL ESOPHAGUS EXTENDING INTO THE SUPERIOR MEDIASTINUM: VALUE OF A RIGHT-SIDED COMBINED THORACOABDOMINAL APPROACH. JERE W. LORD JR. and S. ARTHUR LOCALIO, New York State J. Med. **49**: 2559 (Nov. 1) 1949.

This case is reported from the department of surgery, New York University-Bellevue Medical Center, Postgraduate Medical School and University Hospital. A 56 year old man was admitted for obstruction to swallowing, the cause of which was located by laryngoscopy at 3 cm. below the hypopharynx. Biopsy disclosed a squamous cell carcinoma. A technic was devised whereby the thoracic end of the esophagus and the cardiac end of the stomach could be mobilized and joined after removal of the malignant growth. The detailed procedure cannot be described here.

Unfortunately, the patient died on the fourth day from atelectasis and bronchopneumonia, but, in retrospect, this complication might have been avoided. In future instances tracheostomy at the completion of the anastomosis, with frequent

suction to remove accumulated secretions, is proposed. Although this patient sought aid within four weeks of the beginning of his symptoms, there was extension of the growth beyond the esophagus into the trachea at one point. The authors recommend this right-sided thoracoabdominal approach. No difficulty was encountered on drawing the stomach into the cervical wound. There was surgical success, since the patient reacted favorably up to the time of the complicating pulmonary condition. How to avoid the complication is a problem which must be solved before the next such undertaking. Technical difficulties seem to have been decidedly and surprisingly few. I. W. VOORHEES, New York.

SUPERFICIAL LOBECTOMY AND TOTAL PAROTIDECTOMY WITH PRESERVATION OF THE FACIAL NERVE IN THE TREATMENT OF PAROTID TUMORS. D. STATE, Surg., Gynec. & Obst. **89**:237 (Aug.) 1949.

It is suggested that the high recurrence rate following excision of parotid tumors is due primarily to inadequate excision of the tumor. The lack of a true capsule and the myxomatous tumor tissue make it difficult to determine the true extent of the neoplasm. Fear of injury to the facial nerve is also responsible for inadequate conservative excision of parotid tumors. The author uses an excision which extends vertically from the zygoma to a fingerbreadth below the angle of the mandible and then forward to the posterior border of the submaxillary gland. The skin flap is raised and dissected anteriorly until the anterior border of the parotid body and the parotid duct are visible. After defining the anterior border of the gland, the dissection is carried posteriorly between it and the facial nerve. When the isthmus of the gland is reached, the site of the bifurcation of the main trunk of the nerve can be identified and thus avoided. By transection of the isthmus, the superficial portion of the gland is removed. The deeper portion may be removed by then elevating the facial nerve with a hook and dissecting the gland away from the main stem of the nerve. Radical excision of the superficial portion of a parotid gland or a total parotidectomy should be considered for all solid tumors of the gland; these neoplasms should be assumed to be malignant until proved otherwise. FRIEDBERG, Chicago.

GABRIEL FALLOPIO: ONE OF THE 16TH CENTURY FOUNDERS OF MODERN ANATOMY; ALSO DISTINGUISHED PHYSICIAN AND SURGEON, 1523-1562. WALTER A. WELLS, Laryngoscope **58**:33 (Jan.) 1948.

Herophilus and not Fallopio discovered and described the organs now known as the fallopian tubes, but Fallopio discovered many other important anatomic parts, including Poupart's ligament, the fallopian canal, or aqueduct and the fallopian hiatus. He discovered much about the female pelvis, the temporal bone and several important nerves. He was an authority on syphilis and an able botanist, internist and surgeon.

Not much is known of Fallopio's personal life, but evidence exists that indicates it was estimable. HITSCHLER, Philadelphia.

RHINOLOGY IN CHILDREN: RÉSUMÉ OF AND COMMENTS ON THE LITERATURE FOR 1948. D. E. S. WISHART, Laryngoscope **59**:929 (Sept.) 1949.

Wishart's excellent review includes such subjects as head colds, complications of sinus disease, allergy, irradiation, physiology, tonsils and adenoids, tonsillectomy and poliomyelitis, fractures of the nose, surgery and new growths. The review (and some of the source articles) should be read in full.

HITSCHLER, Philadelphia.

PROCAINE PENICILLIN SOLUTIONS IN THE TREATMENT OF NASAL POLYPOSIS. SIMON L. RUSKIN, Laryngoscope **60**:111 (Jan.) 1950.

The author states that nasal polyps are due to a combination of allergy sensitivity and chronic infection. Solutions of procaine penicillin containing 5,000 units or less per cubic centimeter are nonirritating, and when instilled into the sinuses remain there several days. The penicillin has a bactericidal action and the procaine an antihistaminic action. Sixty patients with polyposis thus treated showed excellent results.

HITSCHLER, Philadelphia.

SKIN TEST WITH TISSUE PROTEINS IN DIAGNOSIS OF FOCAL INFECTION. D. F. DEVANNA, Oto-rino-laring. ital. **15**:326, 1947.

The work in this report consisted in determining whether, in patients affected with chronic tonsillitis and secondary diseases caused by focal infection, a general state of sensitization to proteins produced by the tonsils could be demonstrated; and whether there is no sign of sensitization if a simple hypertrophy or chronic tonsillitis is present without signs of focal infection.

The research concerned 45 patients on whom tonsillectomy had been performed. The antigen was prepared from each individual patient's tonsils, and the skin test was given.

From the results obtained the following conclusions may be drawn. In 76 per cent of the cases with focal infection, general sensitization was evident. The test may be considered of diagnostic value and may be resorted to in doubtful cases for determining the indications for surgical procedure.

MELUCCI, Philadelphia.

MUSCULAR IDIOPATHIC ESOPHAGEAL HYPERTROPHY. C. JEMMI, Oto-rino-laring. ital. **15**: 348, 1947.

The few cases of the described condition recorded in the literature have been detected at autopsy. The condition is symptomless, and the etiology and pathogenesis are undetermined. In the case presented here the thickness of the esophageal wall is as much as 8 to 9 mm. in places; epithelial tissue and connective layers are unmodified. The bulk of the hypertrophy involves the circular muscular layer; the thickness of the longitudinal external layer is increased but little. The dimensions of the muscular cells are normal, but the number of them is tremendously increased. The literature reports cases associated with cardiac hypertrophy and dilatation, pyloric hypertrophy with gastric dilatation, renal sclerosis, chronic bronchitis or emphysema. In this case there was associated prostatic and pyloric hypertrophy.

MELUCCI, Philadelphia.

ENDOSCOPIC STUDY OF BRONCHIAL ASTHMA AND ITS TREATMENT. VASSALLO DE MUMBERT, Bol. espanol otorrhinolaryng. **11**:39 (Jan.-Feb.) 1949.

The author discusses bronchial asthma from the endoscopist's point of view and describes many of the pathologic findings. He classifies 324 patients into two groups. The condition in the larger group is definitely due to bacterial organisms, and that in the small group is due to many allergies. In his series of cases, 38 patients had a single organism as a causative factor and 197 had a mixture of many organisms. He believes that bronchoscopy should be undertaken in every asthmatic patient. Secretions can be aspirated, cultures can be made from which vaccines

can be administered and, finally, local medication applied, particularly the insufflation of penicillin powder directly to the diseased areas. Two hundred and ten patients were completely relieved of all asthmatic symptoms after a period of one to three years.

PERSKY, Philadelphia.

PNEUMOCOCCIC OTITIC MENINGITIS. ARTURO TELLO T. and MARCOS COHN B., Rev. otorrhinolaryng. **9**:53 (June) 1949.

In surveying a group of 53 cases of pneumococcic meningitis of various foci, Tello and Cohn found that an acute otitis media was one of the most frequent sources of origin. Difficulty in early diagnosis was a problem in most of the cases. This difficulty was probably due to the insidious nature of the pneumococcus organism. The meningeal picture is often inconclusive, atypical and not characteristic until an advanced pathologic process is present in the meninges.

The authors emphasize one symptom—an intensive fronto-occipital headache accompanying acute otitis media. If a lumbar puncture is made at this time, significant changes in the spinal fluid can be observed. The surgical appearance of the mastoid process invariably shows a pronounced disproportion between the scant local symptomatology and the extent of involvement of the temporal bone. They also observed that the Pneumococcus produces a proliferative lesion of the mucous membrane of the middle ear and the mastoid cells. Cultures made of this tissue usually yield the organism.

PERSKY, Philadelphia.

CONTRIBUTION TO THE ETIOLOGY, PREVENTION, AND TREATMENT OF HEMORRHAGES IN THE FIELD OF RHINOLARYNGOLGY. HARRY NEIVERT, LEO A. PIRK and RECHA ENGELBERG, Pract. oto-rhino-laryng. **11**:177, 1949.

The authors discuss hemorrhage in otolaryngologic cases and in a comprehensive study emphasize some of the factors which may be conducive to late tonsillar hemorrhage. They stress the point that many of these hemorrhages are secondary to administration of salicylic acid. Salicylates seem to produce a hypoprothrombinemia.

With the administration of vitamin K, the authors found that secondary bleeding was reduced to 1.8 per cent. Results almost similar in nature were obtained by the use of ascorbic acid preoperatively and postoperatively, for it was found that hemorrhages occurred five times as frequently in patients who were deficient in vitamin C as in those with normal levels. Vitamin C was also shown to prevent salicylate-induced hypoprothrombinemia. In conclusion, they state that vitamin K and ascorbic acid both have a place in the prevention of secondary tonsillar bleeding and in the treatment of nasal hemorrhage.

PERSKY, Philadelphia.

NEURALGIAS OF THE SKULL AND THE FACE. O. GODIN, Acta oto-rhino-laryng. bel. **3**:10, 1949.

This report was presented to the Congress of the Belgian Society of Otolaryngology, in July 1949. It comprises 21 chapters dealing with anatomy and physiology of the vessels and nerves of the head (for which the work of Wolff and Penfield is prominently cited), symptomatology, diagnosis and treatment of different types of headaches and facial neuralgias. Following are the conclusions arrived at in this work.

I. The present anatomic and physiologic data indicate that the old theory of "vacuum sinus" and reflex rhinogenous headache did not repose on rational

physiologic basis. The pains referred to the nose and the sinuses are oftenest vascular pains of the extracranial or intracranial vessels caused by circulatory changes (spasm, congestion). The favorable results reported from intranasal and sinus operations appear to be due to circulatory changes produced in the region of the external carotid branches, spreading to other branches of this vessel.

2. The physiopathology of trigeminal neuralgia seems to be a spasm of the branches of the cerebral arteries. The effective treatment is surgical.

3. Migraine is caused in its first phase by constriction of the branches of cerebral arteries, causing visual disturbances, and in the second phase by vasodilatation of the external carotid branches, causing the headache, hence the favorable effect of ergot compounds.

4. Several types of headache are classified in one group as having a common vascular origin: histaminic headache of Horton, syndrome of sphenopalatine ganglion of Sluder, vidian nerve syndrome of Vail, syndrome of the nasal nerve of Charlin, genicular neuralgia of Hunt and the hyperemia headache of Alajuanine.

5. Vasodilatation of the cerebral arteries may cause headache only if the arteries of the base of the skull are involved. This involvement occurs in headache due to histamine injection, an excess of carbon dioxide, carbon monoxide, nitrites and alcohol intake, fever and infections.

6. In hypertensive vascular disease the headache seems to be caused by vasodilatation of the branches of the external carotid, in increased intracranial pressure, and by traction on the venous sinuses due to cerebral edema.

7. Post-traumatic and commotio headache has a mixed background: psychic, vascular and organic.

8. The psychogenic headache is of two types: the first, in a psychopathic person, requires psychiatric treatment; the second, which is commoner, the psychosomatic, occurs in normal psychic background and is due to an imbalance in the autonomic system in connection with frustration, environmental conflicts, sexual and other maladjustments. It has a vascular mechanism (spasm of external carotid branches) and is relieved by procaine (novocain®) infiltrations of these branches (temporal, occipital, frontal) associated with calcium bromide administered intravenously. A 1 per cent solution of procaine without epinephrine should be used. Menopausal headache requires only estrogens.

The author found in cases of functional (psychosomatic) headaches that the venous pressure (as measured with a water manometer on the brachial veins) is below normal and that endocrine imbalance is often present.

HERSON, Chicago.

POLIOMYELITIS AFTER OPERATION ON THE NOSE AND THROAT. J. BÖE and ARNE KÅSS, Tidsskr. norske lægefor. **67**:485 (Sept. 15) 1947.

Of 526 patients with poliomyelitis (401 of whom were children below 15 years of age) treated from 1936 to 1945, manifestations of the bulbar type of the disease occurred two weeks after tonsillectomy in 1 child. This case occurred at a time when poliomyelitis was not prevalent. In that part of Norway, tonsillectomies and other minor operations on the upper respiratory tract are frequent. The authors consider the risk of a patient acquiring poliomyelitis after such operations as almost negligible.

WALLGREN, Stockholm, Sweden [AM. J. DIS. CHILD.].

Book Reviews

Oral and Facial Cancer. By Bernard G. Sarnat, M.D., Professor and Head of the Department of Oral and Maxillofacial Surgery, College of Dentistry, and Clinical Assistant to Professor of Surgery, College of Medicine and Research and Educational Hospital, University of Illinois, Chicago, and Isaac Schour, D.D.S., Ph.D., Sc.D., Coordinator of Cancer Instruction, Professor and Head of the Department of Histology and Associate Dean in Charge of Postgraduate Studies, University of Illinois, College of Dentistry, Chicago. Price, $6. Pp. 300, with 118 figures and 2 plates. The Year Book Publishers, Inc., 200 E. Illinois Street, Chicago, 1950.

The purpose of this book, to serve as a simplified presentation for the general medical and dental practitioner, has been accomplished. The specialist too, perhaps, will find it of some interest.

The text is rather well written, and the illustrations are good, but while the discussion of the present status of cancer research is recent, a book of this nature becomes dated because of the strides that are being made in this field. Also, the section on precancerous oral lesions should have been somewhat more extensive.

The contents are arranged in three main parts: part 1, the cancer problem; part 2, clinical aspects of oral and facial cancer, with relation to carcinoma of the soft tissues, cancer of the jaws and a discussion of cancer in childhood, and part 3, control of oral and facial cancer in regard to the role of the general practitioner and the specialist.

Clínicas y policlínicas otorrinolaringológicas. By Dr. Francisco H. Rivero. Paper. Pp. 311. P. Fernandez y Cia. S. en C., Habana, Cuba, 1950.

This volume is a collection of papers presented by Rivero dealing with many conditions in the upper respiratory tract. Many of these papers have been published previously in various journals or presented before various medical societies. Rivero has attempted to bring the problems discussed up to date. There is no continuity in relationship of the subjects discussed. Some of the subjects are nasal cough; pansinusitis; total rhinoplasty; two cases of mucocele; intranasal syphilis; mycosis of the pharynx; cancer of the larynx; laryngeal tuberculosis; tonsillectomy; hypertrophy of the thymus, and chronic suppurative otitis media. Although each individual subject is particularly well presented, the book as a whole apparently loses its importance.

SPECIAL NOTICE

For the general information of indexers, catalogers and library personnel with reference to the listing of the new title of this journal, we advise that the *Quarterly Cumulative Index Medicus* policy be followed. The *Quarterly Cumulative Index Medicus* beginning with its volume 49 (January-June 1951) will list this journal under the new title, so that the letters A. M. A. will be included as an integral part of the title. For the completion of volume 48 (July-December 1950) the *Quarterly Cumulative Index Medicus* will continue to use the old title, without adding the designation A. M. A.

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Secretary: Dr. Louis H. Clerf, 128 So. 10th St., Philadelphia 7.
Place: White Sulphur Springs, W. Va. Time: May 9-10, 1951.

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Place: White Sulphur Springs, W. Va. Time: May 6-8, 1951 (a. m.).

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Place: Philadelphia. Time: Jan. 12, 1951.
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Place: Miami, Fla. Time: Jan. 17, 1951.
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Place: San Francisco. Time: Jan. 27-28, 1951.

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VOLUME 52
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CONTENTS OF VOLUME 52

JULY 1950. NUMBER 1

PAGE

Esophageal Tumors: Pathology, Diagnosis and Surgical Treatment. P. E. Ireland, M.D., and D. P. Bryce, M.D., F.R.C.S. (C.), Toronto Canada.... 1

Neurofibroma of the Larynx. Samuel J. Pearlman, M.D.; Elmer A. Friedman, M.D., and Max Appel, M.D., Chicago..... 8

The Tympanic Plexus: An Anatomic Study. Samuel Rosen, M. D., New York 15

Osteoma of the Tongue. Robert L. Breckenridge, M.D., and Robert M. Lukens, M.D., Philadelphia..... 19

Evaluation of Newer Drugs in the Treatment of Otitis Externa. E. King Gill, M.D., Corpus Christi, Texas..... 25

Dermatomyositis and Scleroderma: Unusual Causes of Dysphagia. Robert B. Lewy, M.D., Chicago..... 31

House Dust Allergy. Theron G. Randolph, M.D., Chicago..... 40

Otolaryngologic Diseases in Patients with Clinical Avitaminosis. Milton L. Jennes, M.D., Waterbury, Conn..... 58

Congenital Triple Nares. Edgar M. Holmes, M.D., Boston..... 70

Use of Aureomycin in External Otitis: Comparison with Other Drugs Now in Use. William K. Wright, M.D., Houston, Texas..... 74

Treatment of Juvenile Papilloma of the Larynx with Resin of Podophyllum: A Preliminary Report. J. B. Hollingsworth, M.D.; H. W. Kohlmoos, M.D., Oakland, Calif., and R. C., McNaught, M.D., San Francisco..... 82

Accidents of Dental Origin: Oral Prostheses in the Respiratory and Alimentary Systems. Ralph W. Edwards, D.D.S., Kansas City, Mo..... 88

•Ozena: Streptomycin and Nitrofurazone Therapy. William C. Thornell, M.D., Cincinnati 96

Case Reports:
Multiple Lesions in Carcinoma of the Oral Cavity. Claude S. Mumma, M.D., Los Angeles 104

News and Comment 108

Abstracts from Current Literature 109

Society Transactions:
Chicago Laryngological and Otological Society 123

Book Reviews 127

Directory of Otolaryngological Societies 128

AUGUST 1950. NUMBER 2

Ten Year Study of Frontal Sinusitis at the Los Angeles County General Hospital. Fordyce Johnson, M.D., Pasadena, Calif..... 129

Benign Tumors of the Esophagus: Report of a Case of Neurofibroma. Charles F. Engelking, M.D.; M. D. Knight, M.D.; W. H. Brauns, M.D., and L. R. Hershberger, M.D., San Angelo, Texas..... 150

Diseases of the Ear in Relation to Diseases of Other Organs. William L. Wolffheim, M.D., Akron, Ohio 157

Treatment of Empyema of the Sella Turcica of Sphenoid Origin. Joseph R. Rongetti, M.D., and James T. Daniels, M.D., New York..... 166

Primary Carcinoma of the Trachea. M. Reese Guttman, M.D.; Marshall U. Simon, M.D., and Charles B. Nitka, M.D., Chicago..... 172

Psychosomatic Aspects of Surgical Treatment of Otosclerosis. Robert Henner, M.D., Chicago 177

Unusual Nasal Tumors: Report of Two Cases. P. J. Gerity, M.D., Great Falls, Mont. 188

Etiology of Otomycosis. Leanor D. Haley, Ph.D., Durham, N. C.:
I. Mycologic Flora of the Ear..... 202
II. Bacterial Flora of the Ear..... 208

AUGUST—*Continued*

PAGE

III. Observations on Attempts to Induce Otomycosis in Rabbits 214
IV. Clinical Observations 220

Double Malignant Tumor Associated with Pulmonary Tuberculosis and Esophagobronchial Fistula. J. Gordon Brackett, M.D., and Herbert A. Lautz, M.D., Rochester, N. Y. 225

Tumors of the Face. Delmar F. Weaver, M.D., Detroit 234

Streptomycin in Treatment of Scleroma: Report of Three Cases with Successful Results. Milovan Ćurković, M.D., Zagreb, Yugoslavia 253

Fractures of the Facial Bones. W. Raymond McKenzie, M.D., Baltimore 257

Primary Lymphangioendothelioma of the Nose. Wen-Chih Hung, M.D., Taipeh, Formosa 278

Case Reports:

Attempted Fenestration Operation in a Case of Absence of Lumen of Semicircular Canals. Karsten Kettel, M.D., Hillerød, Denmark 284

Progress in Otolaryngology. Summary of the Bibliographic Material Available in the Field of Otolaryngology for 1948:

Peroral Endoscopy. F. J. Putney, M.D.; J. J. O'Keefe, M.D., and L. H. Clerf, M.D., Philadelphia 288

News and Comment 320

Book Reviews 320

Directory of Otolaryngologic Societies 322

SEPTEMBER 1950. NUMBER 3

Mechanism of Suffocation in Spinobulbar Poliomyelitis and Experiences with Operative Treatment. Arne Sjöberg, M.D., Stockholm, Sweden 323

Tracheotomy in Poliomyelitis. Robert J. Strobel, M.D., and Norton Canfield, M.D., New Haven, Conn. 341

Arteriovenous Aneurysm of the Internal Carotid Artery in the Cavernous Sinus. Louis M. Freedman, M.D., Boston 351

Effect of Autonomic Denervation on Nasal Mucosa: Interruption of Sympathetic and Parasympathetic Fibers. Arthur F. Millonig, M.D.; Harold E. Harris, M.D., and W. James Gardner, M.D., Cleveland 359

Hyaluronidase in Rhinoplasty. Maurice H. Cottle, M.D.; J. Allan Weiss, M.D.; Edward Pottorff, M.D., and Emanuel Herzon, M.D., Chicago 369

Ménière's Syndrome: A Proposed Classification and Differential Diagnosis. Burech Rachlis, M.D., F.I.C.S., Philadelphia 373

Syndrome of Bilateral Vestibular Paralysis and Its Occurrence from Streptomycin Therapy. Captain Page Northington (MC), U.S.N. 380

A Study of the Deafness Heritage in Otosclerosis. Samuel J. Kopetzky, M.D., New York 397

Chemical Burns of the Oral Cavity and Esophagus. Angel V. Stumboff, M.D., Chicago 419

Progress in Otolaryngology. Summaries of the Bibliographic Material Available in the Field of Otolaryngology:

The Paranasal Sinuses. Samuel Salinger, M.D., Chicago 427

Abstracts from Current Literature 496

Society Transactions:

Second International Audiology Conference 502

Book Reviews 511

Directory of Otolaryngologic Societies 512

OCTOBER 1950. NUMBER 4

Evaluation of the Operative Indications and Results in the Fenestration Operation for Otosclerosis. Edward H. Campbell, M.D., Philadelphia ... 513

The Maginot Line of Otology: Dependence on Antibiotics in Suppurative Middle Ear Disease; Facts and Fancies. Francis L. Lederer, M.D., Chicago 533

OCTOBER—*Continued*

PAGE

Antrochoanal Polyp. Walter E. Heck, M.D.; Olav E. Hallberg, M.D., and Henry L. Williams, M.D., Rochester, Minn. 538

N-Butanol: Its Use in Control of Postoperative Pain in Otorhinolaryngological Surgery. Bernard Welt, M.D., Brooklyn. 549

Malar Bone Fractures. M. M. Hipskind, M.D., Chicago 565

Nasal Cavity of the Guinea Pig in Experimental Work. George Kelemen, M.D., Boston 579

Management of Nasal Allergy. Solomon Slepian, M.D., Brooklyn. 597

Fixation of the Vocal Cords in Acromegaly. John K. Grotting, M.D., and John deJ. Pemberton, M.D., Rochester, Minn. 608

Fenestration by Cold Fracture Method: Preliminary Report of an Improved Technic. Samuel Rosen, M.D., New York. 618

Surgical and Allied Diseases of the Salivary Glands and Ducts. William G. Abel, M.D., New York. 622

Case Reports:

Aseptic Lateral Sinus Thrombosis: Report of a Case. Goodlatte B. Gilmore, M.D., and Alan Austin Scheer, M.D., New York. 639

Death Following Tonsillectomy. G. W. Taylor, M.D., Philadelphia. 642

Congenital Malformation of the External Ear and Atresia of the Auditory Canal. George B. Ferguson, M.D., and Carl N. Patterson, M.D., Durham, N. C. 646

Meningitis, Pneumococcic Type 3; Mastoidectomy, Recovery. Benjamin H. Shuster, M.D., and William Gordon, M.D., Philadelphia 650

News and Comment. 654

Abstracts from Current Literature 655

Book Reviews 667

Directory of Otolaryngological Societies 670

NOVEMBER 1950. NUMBER 5

Corrective Surgery of Nasal Deviations. John Marquis Converse, M.D., New York. 671

Monostotic Fibrous Dysplasia of the Mastoid and the Temporal Bone. Charles E. Towson, M.D., Philadelphia. 709

Normal Development of the Ear and Its Mechanics. Franz Altmann, M.D., New York. 725

Tuberculous Retropharyngeal Abscess Treated by Surgery and Streptomycin. Morley T. Smith, M.D., New Rochelle, N. Y. 767

Case Reports:

Deafness and Kernicterus. Roy N. Barnett, M.D., and Clifford F. Ryder, M.D., Norwalk, Conn. 771

Abscess of the Nasal Septum and Orbital Cellulitis in an Infant. G. Douglas Hayden, M.D.; Peter N. Pastore, M.D., and Karl Kundert, M.D., Richmond, Va. 773

Retropharyngeal Abscess. Frank L. Bryant, M.D., Shreveport, La. 776

Intratemporal Sarcoma of the Facial Nerve. Karsten Kettel, M.D., Hillerød, Denmark 778

Recruitment Phenomenon in Craniocerebral Trauma: Report of Representative Cases. Maurice Saltzman, M.D., Philadelphia. 782

Clinical Notes; New Instruments and Technics:

Irrigation of the Maxillary Sinus Through the Natural Opening with a New Irrigator. Max Unger, M.D., New York. 786

An Endaural Retractor with Temporal Muscle Blade. Robert Henner, M.D., Chicago 792

A Useful Face Mask for Endoscopic Examinations. Bernard Jaffe, M.D., Walla Walla, Wash. 793

NOVEMBER—*Continued*

PAGE

Progress in Otolaryngology. Summaries of the Bibliographic Material Available in the Field of Otolaryngology for 1948:
Allergy. E. L. MacQuiddy, M.D., and E. A. Holyoke, M.D., Omaha 794
Abstracts from Current Literature 829
Directory of Otolaryngological Societies 842

DECEMBER 1950. NUMBER 6.

Symposium on the Histology and Pathogenesis of Otosclerosis:
Fissular Region of the Otic Capsule in Relation to Otosclerosis. Barry J. Anson, Ph.D. (Med. Sc.), Chicago 843
Incidence, Location and Extent of Otosclerotic Lesions. Stacy R. Guild, Ph.D., Baltimore 848
Otosclerosis: Hypothesis of Its Origin and Progress. Dorothy Wolff, Ph.D., New York 853
Influence of Systemic and Local Factors on the Development of Otosclerosis. J. R. Lindsay, M.D., Chicago 868
Postnatal Rebuilding and Otosclerotic Bone Formation in the Region of the Otic Capsule. T. H. Bast, Ph.D., Madison, Wis. 882
Critical Evaluation of Antihistaminic Drugs in the Common Cold. Noah D. Fabricant, M.D., Chicago 888
Classification of Chronic Diseases of Nose and Accessory Sinuses. Noah Fox, M.D., and Leonard M. Niles, M.D., Chicago 900
Streptomycin Therapy in Oral Tuberculosis. H. Oppenheim, M.D.; C. S. Livingston, M.D.; J. W. Nixon, D.D.S., and C. D. Miller, D.M.D., Outwood, Ky. 910
Tympanomeatal Membrane in the Fenestration Operation: Its Relation to Bony Closure of the Fenestra. Samuel Rosen, M.D., New York 930
Use of Vinegar as an Antibiotic in the Treatment of Chronic Middle Ear Disease. Irving L. Ochs, M.D., Annapolis, Md. 935
Case Reports:
Rhabdomyosarcoma of the Maxillary Antrum: Seven Year Survival Following Surgical Excision and Radiation Therapy. Peter N. Pastore, M.D.; Philip F. Sahyoun, M.D., and Frederick B. Mandeville, M.D., Richmond, Va. 942
Clinical Notes:
Histamine Treatment of Bell's Palsy: Report of Five Cases. G. L. Loomis, M.D., Winona, Minn. 948
An Operation for Relief of Stenosis of the Stoma Following Total Laryngectomy. John Marquis Converse, M.D., New York 950
A Skin Resistance Audiometer. John A. Kirchner, M.D., New Haven, Conn., and Harry H. Haggart, M.D., Cincinnati 952
Infraction of the Inferior Turbinates. Otto L. E. De Raadt, M.D. (Leiden), Tjimahi, Java 956
Tooth Guard for Use with Davis Mouth Gag. Ralph S. Lloyd, D.D.S., and Rheim M. Jones, M.D., Baltimore 960
A New Nasal Splint. Irving B. Goldman, M.D., New York 962
Progress in Otolaryngology. Summaries of the Bibliographic Material Available in the Field of Otolaryngology for 1947 and 1948:
Plastic Surgery. Lyndon A. Peer, M.D.; John C. Walker, M.D., and John Van Duyn, M.D., Newark, N. J. 964
Special Reports:
Hearing Aids Accepted by the Council on Physical Medicine and Rehabilitation, the American Medical Association, as of November 1, 1950 993
Abstracts from Current Literature 998
Book Reviews 1011
Special Notice 1011
Directory of Otolaryngological Societies 1012
Contents of Volume 52 i

(Advertisement)

PSYCHOSOMATIC ASPECTS OF HEARING DEFECTS

Rehabilitation of the Hard-of-Hearing

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A defect in hearing initiates emotional and practical problems which profoundly affect the individual's attitude toward himself and society. In turn his handicap influences the way people act toward him. Some aspects of this complex problem are briefly discussed in this time-saving refresher for the busy practitioner.

HELP TO REBUILD DREAMS

Whether a hearing defect is the sudden result of trauma or develops gradually, the realization that the handicap is *permanent* and may become greater, strikes the victim with a shattering impact. The young make adjustment more easily [1] but the older patient faced, as he may be, with a handicap which makes it impossible to continue with the profession in which, after years of struggle, he has established himself, faced with the necessity of finding a new way to maintain economic security, has one of the most difficult of all problems of readjustment.

Having assured himself that everything possible, both medically and surgically, has been done to help his hard-of-hearing patient, the physician must then turn to the problem of rehabilitation. Both psychologic and psychiatric investigation may be required.[2] The physician must help his patient to rebuild his dreams. It may be helpful to point to the famous deaf, to Beethoven, Edison, and Bernard Baruch, for example, who have continued their satisfactory careers in spite of the handicaps of extreme hearing disability.

Recognition of the psychosomatic aspects of hearing defects and of the means for relieving them are the touchstones by which the physician helps his patient to adjust himself to the limited world in which he must live. Otherwise he is dealing with an "unredeemed human tragedy." [3]

THE HARD-OF-HEARING IS AN INDIVIDUAL

Each patient with a hearing defect must be treated as an individual. No two patients are alike. Each has a different pattern of hearing loss. Each has his own environment, which shapes his attitudes toward life, his personality. Understanding all of these, discovering the aptitudes and possibilities of the patient, perhaps even unearthing new talents,[4] these are all means the physician can use in rehabilitating the acoustically impaired.

Rehabilitation starts where medicine and surgery end. Morrissett [2] has said that "a deafened patient who is not adjusted to the life he must live represents a failure of treatment." The motto of the Reading Hospital rehabilitation clinic for the aurally handicapped reads:

> "any deaf or hard-of-hearing person may represent a phenomenal surgical or medical success, but if he is not equipped to fit readily into the pattern of his civilian life he will represent a failure as far as vocational rehabilitation is concerned." [2]

COORDINATE ACTIVITIES FOR REHABILITATION

Few hard-of-hearing persons have any idea of the means available to help them rehabilitate themselves. They should be made familiar with them.[3] Outside of federal institutions there are not available facilities for mass rehabilitation of the hard-of-hearing.[2] The burden, then, rests on the shoulders of the individual physician.

Rehabilitation requires a program which combines medical care, psychological adjustment and vocational guidance. The patient must be informed of the possibility of improving his hearing by the use of mechanical devices, of the value of lip reading. He must be encouraged to mingle with people, to talk with them. "The most important phase of the aural rehabilitation program is perhaps the coordination of all its activities," Morrissett [2] states. "The patient is not left to his own devices to secure a hearing aid, to learn how to use it, to learn lip reading and to use whatever else he may wish to do or may not wish to do."

Too often the hard-of-hearing believes that the purchase of a hearing aid will solve all his problems. The physician recognizes that his patient must learn to use the aid by systematic auditory training.[5] "Cerebral reorientation to speech" as transmitted through a hearing aid can thus be improved.[6]

A useful supplement to rehabilitation is keeping a diary. The patient should note the new sounds he hears as well as the old sounds he hears more distinctly. The doctor, too, will find this diary useful in evaluating his patient's progress.[6,7]

Above all, "the patient must be encouraged out of the deaf-class-consciousness. He must be given the will to hear, must have faith that the treatment is going to improve his function." [7] Both the patient and the doctor need "utmost patience." [7]

With proper coordination of the various parts of the rehabilitation program, with sympathetic understanding on the physician's part, there is no reason why the hard-of-hearing patient should not continue to lead a useful and happy life.

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5. Stevenson, R. S.: Recent Advances in Oto-Laryngology. J. & A. Churchill, Ltd., London, 2nd ed. 1949, pp. 81-82.
6. Barnett, H. N.: M. Press **216**: 174-175 (Sept. 4) 1946.
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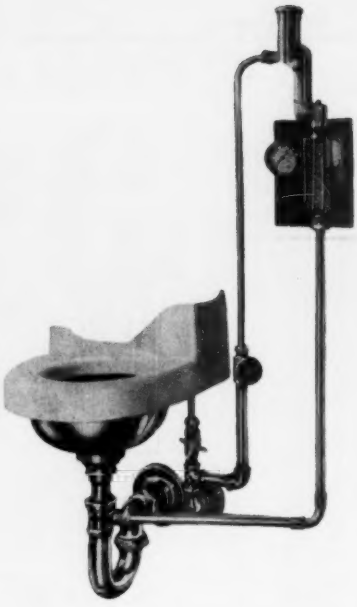

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1. Waldbott, G. L.: Management of Allergic Eczema. Am. Pract. 4:59-63 (Oct.), 1949.

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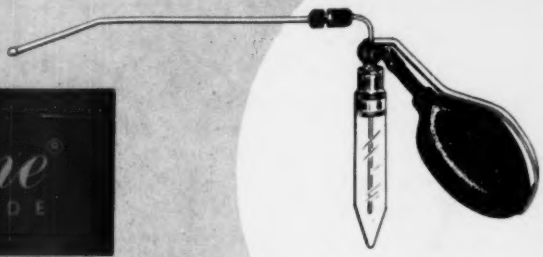

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1. Carabelli, A.: *Diseases of the Chest*, 15:532, May, 1949.
2. George P. Pilling & Sons, Company, 3451 Walnut Street, Philadelphia 4, Pa.

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